THE
LARYNGOSCOPE.

VOL. XVII. ST. LOUIS, MO., NOVEMBER, 1907. No. 11.

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

VISUAL DISTURBANCES SHOWING A CAUSAL RELATION TO DISEASE IN THE SPHENOIDAL SINUSES. WITH THE REPORT OF A CASE, TOGETHER WITH CORONAL AND SAGITTAL SECTIONS DEMONSTRATING THE RELATIONS EXISTING BETWEEN THESE CAVITIES.*

BY JOHN W. MURPHY, A.M., M.D., CINCINNATI.

It is not my intention, in this paper, to go into the history of suppuration in the posterior ethmoid and sphenoid cells, as since the first report of suppuration in these sinuses in 1872,[1] to the present time the literature is prolific on this subject. This is especially the case since 1885, when Schaeffer began to call our attention to the symptoms and treatment of the disease in the living. Nor do I wish to go into an analysis of all of the symptoms which may manifest themselves in disease of the sphenoid and ethmoid cells. I simply wish to confine myself to the report of a case which I have met with recently, in which the eye symptoms were pronounced, and the causal connection seemed traceable to disease in the spenoidal cells. This causal connection was also confirmed by necropsy. I also wish to exhibit some specimens which were made during my investigation into this subject, and which will illustrate the close relation existing between the contents of the orbital cavity and the ethmoid and sphenoid cells.

The intimate relation existing between these two cavities, and what effect disease in the one can have upon the other, must always be a question of much importance to the rhinologist and ophthalmologist.

* Candidate's Thesis. Read before the Thirteenth Annual Meeting of the American Laryngological, Rhinological and Otological Society, New York City, May 30, 31 and June 1, 1907.

That these cavities are more often the seat of disease than we suspect is constantly being forced upon us by the revelations of the post-mortem room. The suppuration is usually caused by the severer forms of acute rhinitis, while certain forms of influenza seem especially prone to attack the sphenoid cells. There seems to be no question but what sphenoidal and ethmoidal suppurations are more frequently met with now than formerly. In the European clinics of Chiari and Lichtwitz, only about 2 per cent of the cases show sphenoidal suppuration.

In the examination of 600 cases in the Massachusett's General Hospital, Rogers,[3] found these cells diseased in 5 per cent of the cases. My experience leads me to believe this higher per cent is more nearly correct for our country.

That the sphenoid is rarely diseased alone but nearly always in conjunction with one or more of the other accessory cavities of the nose seems to be the experience of all observers.

The difficulty of inspecting or even probing the normal sphenoidal opening, which is situated just above the posterior end of the middle turbinate, no doubt accounts for the low percentage of recorded cases. In a number of cut sections in my collection the anatomical relation of the posterior end of the middle turbinate, or the superior turbinate and ethmoid bulla, render the opening exceedingly difficult, if not impossible, to enter with the probe during life. The average depth of the opening is about 7.5 cm. from the anterior nasal opening, and the probe passes in at an angle of 35 degrees with a line perpendicular to the nasal opening. As you can see upon the cut section, the anterior wall of the sphenoid forms the upper edge of the choanae and by keeping this point in mind, as suggested by Gruenwald[4], the normal opening may more readily be located by the probe.

From the close connection existing between the optic nerves and the cavernous sinuses, with the sphenoidal sinuses, separated as they are by such thin bony walls, or even by a membrane only, it seems strange that disease in these cells is not more often manifested by pronounced eye symptoms . I think we are beginning to recognize more and more that many cases of retro-bulbar neuritis of obscure cause may have their origin in a suppurating sphenoidal cell. This is especially true if the optic neuritis is unilateral in character. Since I have been investigating the subject more closely, I feel quite certain that a causal relation did exist between

several obscure cases of optic neuritis, seen in the past, and a sphenoidal suppuration, but I did not connect them at the time.

Dr. C. R. Holmes,[5] has recorded a number of such observations, in which there could be no question of the causal connection of the eye symptoms and a suppurating sphenoidal cell.

The fact that the veins of the orbit discharge the greater part of their blood through the ophthalmic veins into the cavernous sinuses, which is only separated from the sphenoidal cells by the thinnest of bony walls, or even by a membrane only explains why an inflammation in the one can easily extend to the other and produce all of the symptoms of a retro-bulbar neuritis.

I feel that our knowledge with regard to the network of veins and lymph passages of these cavities is far from perfect, and I feel certain that the more we investigate the relation, the more often we will be able to find a causal connection between certain eye symptoms and diseased sphenoidal or ethmoidal cells.

The number of such cases reported each year is far in excess of those of the past, and I hardly think it can be due to an increased zeal in diagnosis, but rather to a failure on our part to make a diagnosis. As Gruenwald[6], says, "Ophthalmic troubles, such as asthenopia, scotoma, painful irritation of the ophthalmic nerves, and deterioration of writing power as noted by Schaeffer, are explicable by the neighborhood of the orbital and the basal nerves. Pain is usually more violent in acute inflammation, but then it is, on account of radiation to more distant regions, still more uncertain as a diagnostic sign. The same holds good with disease of the ethmoid bone, only where the troubles are usually more severe than in any other cavity, and here, more especially, that disagreeable complication of constant pain, mental depression and intolerance to nervous excitement may be found. Asthenopia is also frequent, and the pain will be referred to the region of the eyes."

In order to get the experiences and opinions of various ophthalmologists as to the causal relation between loss of sight, or optic neuritis, due to disease in the sphenoid and ethmoidal sinuses, Prof. Onodi[7], submitted to a number of them a series of questions on this subject. From their answers you will see that even in the large eye clinics of Europe, a causal relation is not commonly recognized.

Prof. Leber, says: "Although I have turned my attention for many years to the connection between the diseases of the posterior

sinuses of the nose, and those of the organs of sight, I have been able to note very little worthy of record with regard to the sphenoidal sinuses and the posterior ethmoidal cells."

Schmidt-Rimpler: "My experiences afford me no proof that empyema of the sphenoid cavities can of itself cause an affection of the nerves."

Prof. H. Sattler: "Unilateral optic neuritis and optic atrophy are in no way characteristic of disease of the sphenoid and ethmoid cavities."

Prof. Axenfeld: "It is my opinion that diseases of the optic nerves, (neuritis, pressure atrophy) are, if we except actual tumors and perforating cases, much rarer than we might expect in diseases of the sphenoid."

From these reports you can see that the number of recorded cases in which the eye symptoms seemed to have a causal connection with a suppurating sphenoidal sinus, and which were proven at the necropsy, are still comparatively rare.

This, no doubt, can be accounted for by the protection of the sphenoidal bone, whose upper and outer walls are much thicker than the anterior, through which the natural opening for discharge occurs.

As you can see upon the coronal section, through the frozen specimen, in which the cavernous sinus is cut through just in front of the optic chiasm, the contained vessels and nerves, going to or returning from the orbit, are protected by quite a thick bony wall from the sphenoidal sinus. Doubtless in rare cases the protection is not so complete, and it is in these cases that a causal relation may exist between a suppurating sphenoid and certain definite ocular symptoms. When extensive necrosis of the body of the sphenoid is present, then the ocular symptoms may be easily accounted for.

While my patient denied a specific history, yet the extensive destruction of bone found at the time of operation, and later at the post-mortem, was strongly suggestive of a syphilitic origin.

In this case with the eye symptoms coming on so suddenly, following the bony necrosis of the sphenoid, I think there can be no question of the causal relation existing between the two.

CASE REPORT.

Mr. E. H., age 32, came on my service at the Cincinnati Hospital, in October, 1906. He was referred by Dr. W. F. Moss, of Maine-

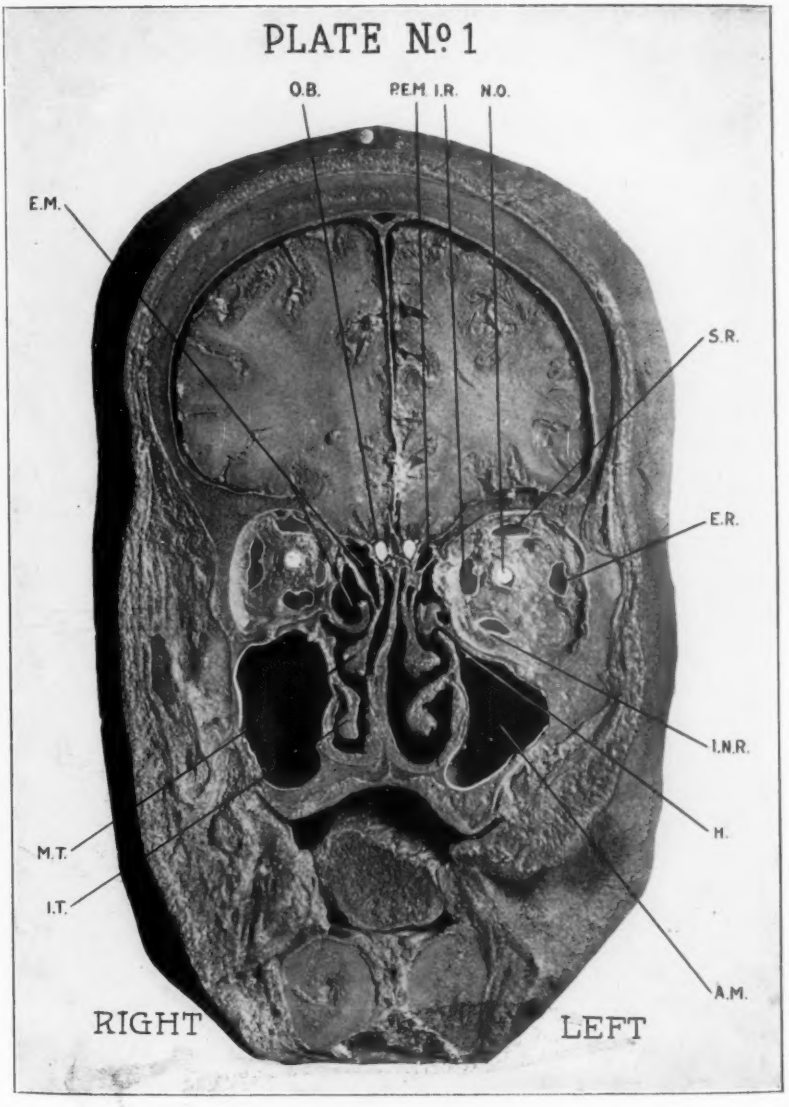


PLATE NO. 1.

Frozen Injected Coronal Section, showing the relation of the posterior ethmoid cells to the orbital and cranial cavities. I. T. Inferior Turbinate. M. T. Middle Turbinate. E. M. Ethmoid Cells. O. B. Olfactory Bulb. P. E. M. Posterior Ethmoid Cell extending over the sphenoid sinus. I. R. Internal Rectus Muscle. N. O. Optic Nerve. S. R. Superior Rectus Muscle. E. R. External Rectus Muscle. H. Hiatus Semilunaris. A. M. Antrum Maxillaris. I. N. R. Inferior Rectus Muscle.

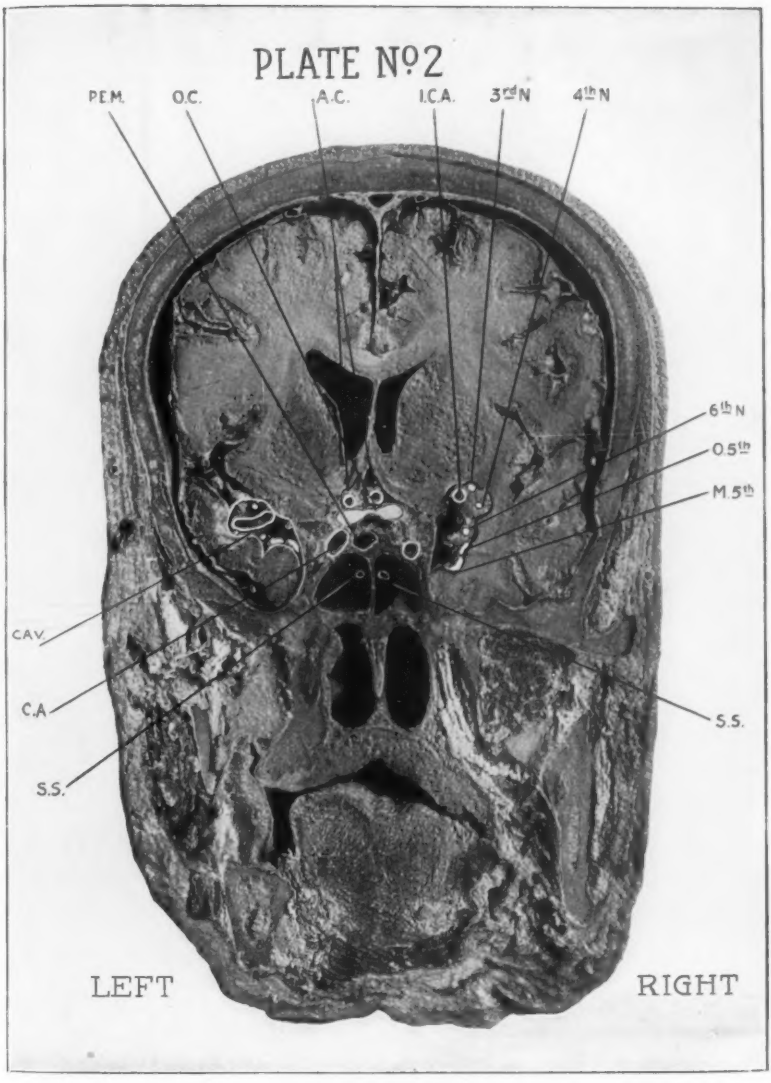


PLATE NO. 2.

Injected Frozen Coronal Section through the optic chiasm and the cavernous sinus, showing the relation of its contained vessels and nerves. S. S. Sinus Sphenoidalis, right and left, showing ostium Sphenoidalis. P. E. M. Posterior Ethmoid Cell extending back over the sinus. O. C. Optic Chiasm. C. A. Carotid Artery in Bony Canal. I. C. A. Internal Carotid Artery in Cavernous Sinus. 3rd N. Third Nerve. 4th N. Fourth Nerve. 6th N. Sixth Nerve. 5th O. Opthalmic Division of Fifth Nerve. 5th M. Sup. Maxillary Division of the Fifth Nerve.

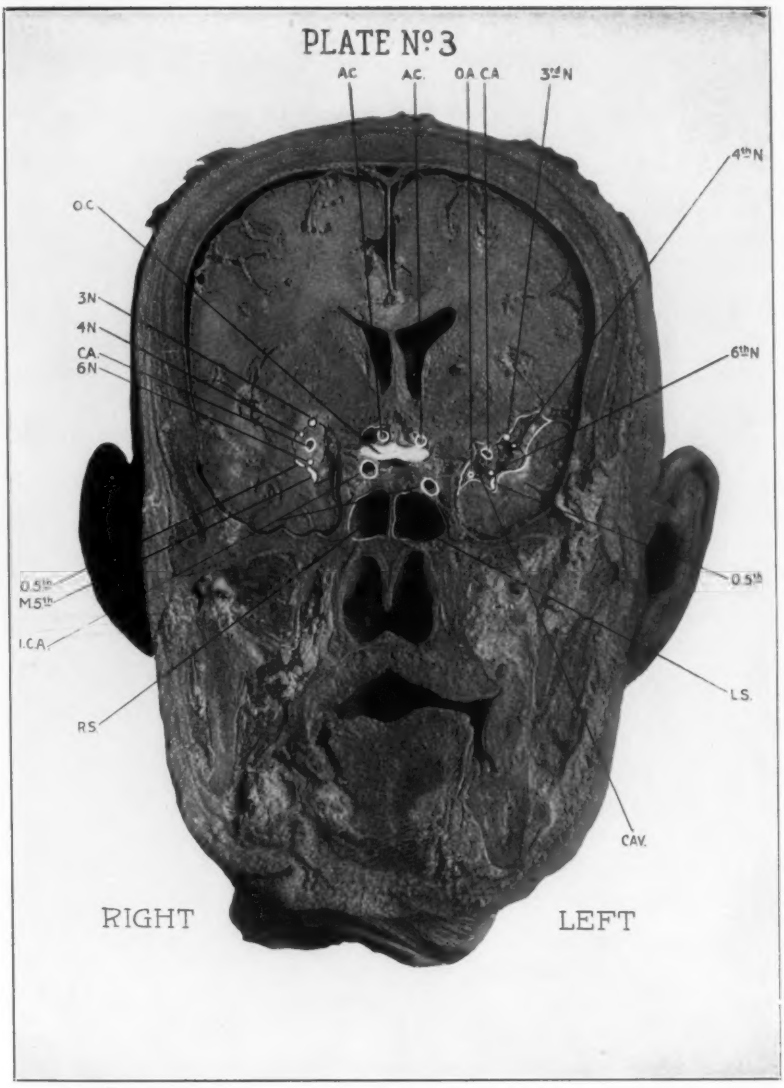


PLATE NO. 3.

Injected Frozen Coronal Section through the Cavernous sinus just back of the optic chiasm, showing the relation of the sinus to the sphenoidal cells and the position of the arteries and nerves in the cavernous sinus. C. A. V. Cavernous Sinus. C. A. Carotid Artery-Internal. 3rd N. Third Nerve. 4th N. Fourth Nerve. 6th N. Sixth Nerve. O. 5th N. Ophthalmic Division of Fifth Nerve. L. S. Left Sphenoid. R. S. Right Sphenoid. I. C. A. Internal Carotid Artery in Bony Canal of Sphenoid. M. 5th. Sup. Maxillary Division of Fifth Nerve. O. C. Optic Chiasm. A. C. Anterior Cerebral Artery, right and left. O. A. Ophthalmic Artery.

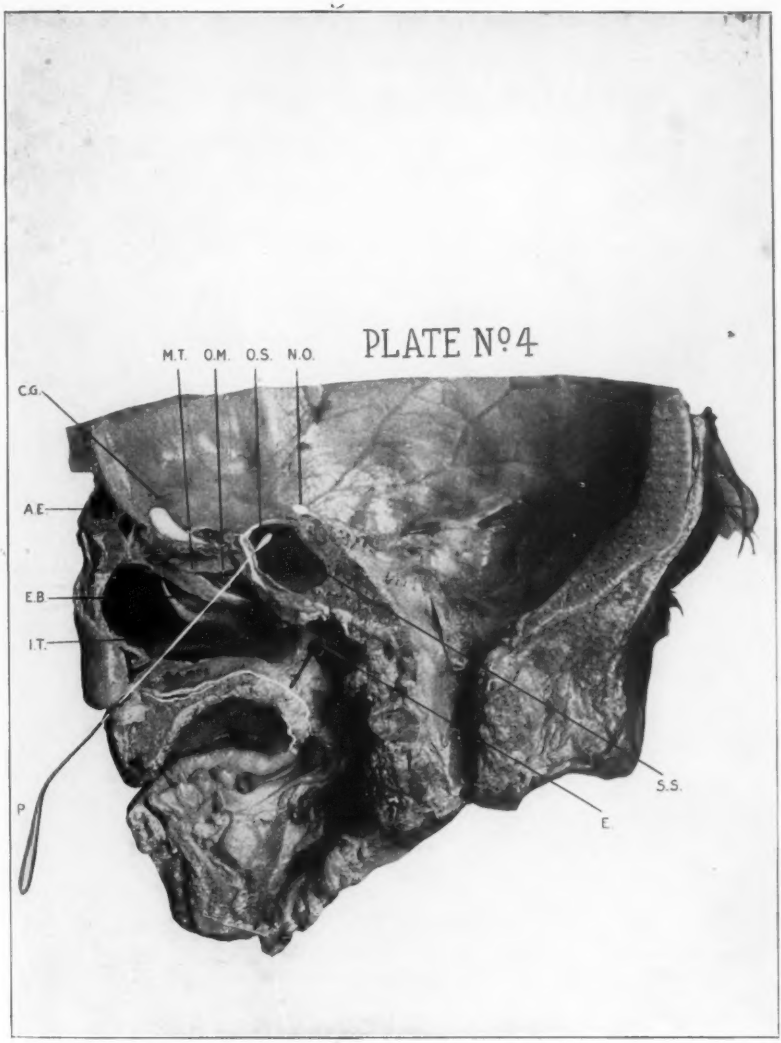


PLATE NO. 4.

Sagittal Section, showing the relation of the sphenoid with the optic nerve. Turbinated bodies atrophied. S. S. Sinus Sphenoidalis 30 m.m. long; 25 m.m. broad; 20 m. m. deep. E. Bougie in Eustachian Tube. N. O. Nervus Opticus. O. S. Ostium Sphenoidalis. O. M. Ostium Maxillaris. M. T. Middle Turbinate atrophied. A. E. Anterior Ethmoid Cell. E. B. Ethmoid Bulla. I. T. Atrophied Inferior Turbinate. P. Probe passing through Ostium Sphenoidalis.

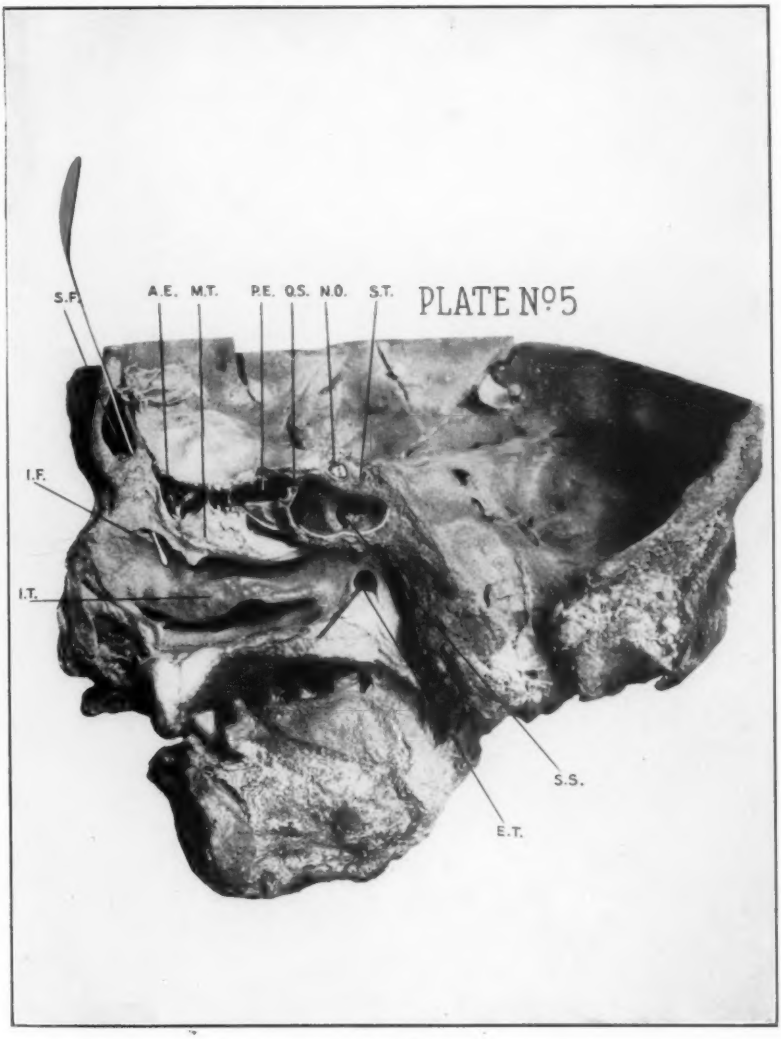


PLATE NO. 5.

Sagittal Section, showing the relation of the sphenoid with the optic nerve and the ethmoids with the frontal sinus. S. S. Sinus Sphenoidalis. E. T. Bougie in Eustachian Canal. S. T. Sella Turcica. N. O. Nervus Opticus. O. S. Ostium Sphenoidalis. P. E. Posterior Ethmoid Cells. M. T. Middle Turbinate. A. E. Anterior Ethmoid Cell. S. F. Sinus Frontalis. I. F. Infundibulum with probe passing from the Frontal Sinus. I. T. Inferior Turbinate.

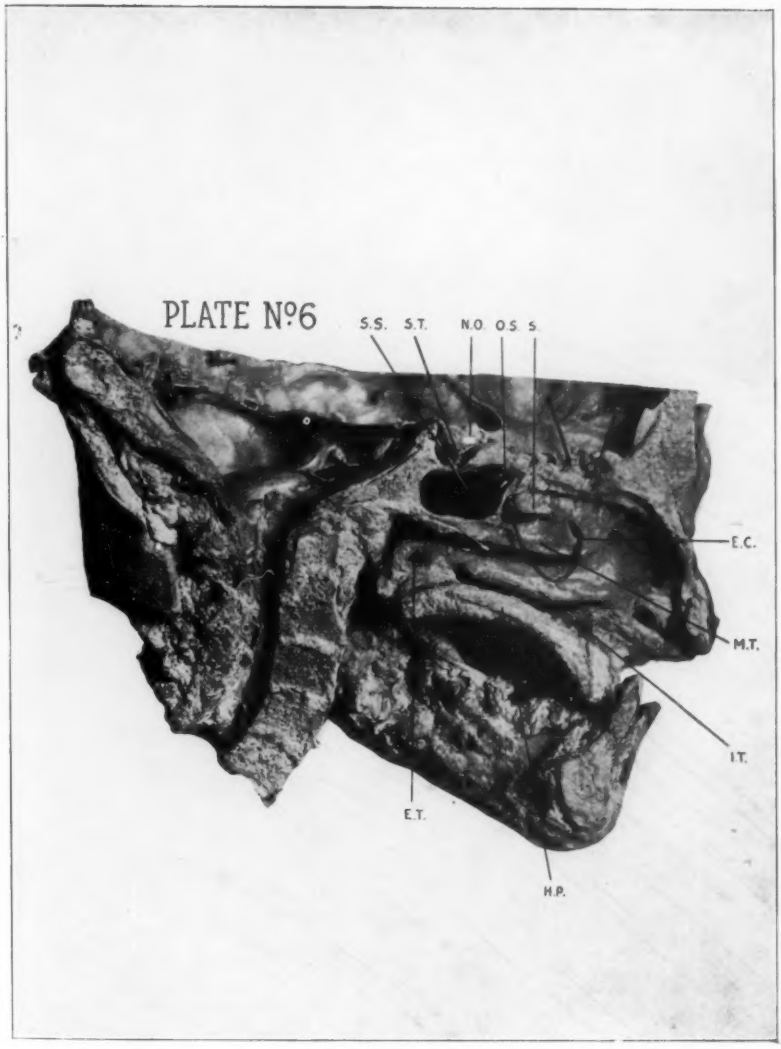


PLATE NO. 6.

Sagittal Section of the left half of the head, showing sphenoid and ethmoid cells. E. T. Eustachian Tube. H. P. Hard Palate. I. T. Inferior Turbinate. M. T. Middle Turbinate. E. C. Ethmoid Cell in Middle Turbinate, with probe passing back into sphenoidal opening. S. Superior Turbinate. O. S. Ostium Sphenoidalis. N. O. Nervus Opticus. S. T. Sella Turcica. S. S. Sphenoidal Sinus.

ville, Ohio, for an empyema of the right accessory cavities of the nose, followed by great mental deterioration, with sudden paralysis of the right external rectus muscle and blindness of the right eye. The doctor states that the patient was apparently well up to three months ago and was able to follow his calling, that of a conductor on a traction line.

Status Praesens:

The patient walks with a staggering gait, so that he has to be assisted. His mental condition is such that he can not give a clear history of his case. There is some exophthalmos of the right eye which turns toward the nasal side from complete paralysis of the external rectus muscle. The position of the eye ball is about 5 m. m. below the horizontal line of the other eye. Vision of the right eye nil. Vision of the left eye about normal. Ophthalmoscopic examination showed almost complete atrophy of the optic disc, more marked on the temporal side. The blood vessels, however, were nearly normal in size and number. This is the last expression of a retro-bulbar neuritis, doubtless secondary to an inflammation in the right posterior ethmoidal and sphenoidal cells. Examination of the left eye showed it to be normal.

Dr. D. T. Vail, saw the case in consultation and verified the ophthalmoscopic examination, and considered the condition due to suppuration in the sphenoidal sinus.

The patient complains of great pain at the occiput and at the base of the brain. There is a most offensive discharge from the right nostril, both anterior and posterior. The probe shows extensive necrosis of the posterior ethmoidal cells and the sphenoid sinus. The left side was not affected. Transillumination showed very dark area over the right maxillary antrum with absence of the pupillary reflex. The left maxillary antrum was clear and the pupillary reflex well marked. There was no difference between the reflex in the right and left frontal, both showing clear. Two X-Ray pictures were taken and both corresponded in every particular with the transillumination test.

Under chloroform anaesthesia the right frontal sinus was opened, to be sure no infection was present, since it seemed hardly possible for any sinus to escape, so extensive was the necrosis on the right side. However, the sinus appeared perfectly healthy and the wound was at once closed. The middle turbinate was removed and the necrotic ethmoid cells broken down. The maxillary antrum was entered through the canine fossa. When the antrum wall

was exposed it was found to be black from the necrotic condition present within the antrum. When the antrum was opened a most sickening odor escaped along with pus and necrosed bone. The nasal wall was removed by the Caldwell-Luc method.

The sphenoid could now be inspected and upon attempting to remove the anterior wall it was found that the whole right half of the sphenoid bone was necrosed and movable. With a finger in the pharynx, this loosened condition could be easily made out. Under the guidance of the finger in the pharynx, alligator forceps were passed in through the opening in the canine fossa, and several large pieces of necrotic bone were removed. Others could be felt but I feared to remove them lest the cavernous sinus might be injured. There was present the most extensive bone destruction I had ever encountered and lead me to believe that the disease must have existed longer than three months, as stated by the patient. It was difficult to see how a patient could exist with such an extensive necrotic condition present.

The cavity was packed with iodoform gauze through the opening in the canine fossa. The patient rallied nicely from the operation and on the following morning said he felt much better than he had for a long time, since the pain at the top of the head and back of the neck was gone. He never complained of the pain afterwards.

For several weeks following the operation his mental condition improved. The wound over the right frontal healed by first intention. Three weeks later he succeeded in tearing the wound open, by constantly picking at it, and the frontal sinus became infected. About this time also, the right ear began to discharge. On the fourth week following the operation a persisting and exhausting diarrhoea developed followed by a diffuse and severe bronchitis and the patient died, December the third, five weeks after the operation.

POST-MORTEM NOTES.

Post-mortem by Dr. Frank Hegner, Pathologist to the Cincinnati Hospital. Adult, male, markedly emaciated, phthisical chest, post-mortem rigidity absent. Above internal angular process of the right eye there is a small wound of a previous operation. Foul odor about the head.

Brain—On removing the calvarium and opening the right frontal sinus, a sero-purulent exudate was found in the latter cavity. This

same inflammatory condition was found in the tissues of the orbital fossa, and with increasing severity to complete necrosis in the right anterior and posterior ethmoidal cells. also the right sphenoidal sinus, and the left posterior ethmoidal sinus. To a less degree in the left anterior ethmoidal cells. The process caused necrosis of the floor of the orbit, and extended directly into the antrum, the posterior and lateral walls of which were completely necrosed. Nothing remained of the sphenoidal sinus excepting debris.

Communication between the right middle cerebral fossa and the pharynx was direct. The right middle and internal ear showed sero-purulent inflammatory changes.

Membranes—Dura in the right middle fossa was markedly thickened and very firmly adherent to the sphenoidal fissure and adjacent bone. Over the body of the right sphenoidal bone this thickened dura formed the only barrier between the brain and the necrotic sphenoidal sinus.

Pia and arachnoid showed over the right hemisphere, anteriorly and inferiorly, marked lepto-meningitis. On section no macroscopic lesion was found in either hemisphere.

BIBLIOGRAPHY.

1. ROUGE. *Union med.* Paris, 1872.
2. SCHAEFFER. Chirurg. Erfahrungen in der Rhinol. und Laryngol., Wiesbaden, 1885.
3. *Boston Med. and Surg. Journ.*, Dec., 1903.
4. Nasal Suppurations, 1901, Gruenwald, page 280.
5. *Arch. Ophth. and Otol.*, N. Y., Vol. xxv, 1896.
6. *Journ. Laryngol.*, London, Nov., 1902.
7. *Journ. Laryngol.*, London Dec., 1904.

4 W. 7th St.

RADIOGRAPHY AND TRANSILLUMINATION IN DIAGNOSIS OF SINUS DISEASE.*

BY JOSEPH C. BECK, MD., CHICAGO.

The value of radiography for diagnostic purposes is so well established at the present time, especially in connection with general surgery, that I feel it is not necessary to take up its origin and progress. However, so far as its application in our specialty is concerned, I must admit that it is very much neglected. The cause, I believe, is the fact that the results thus far obtained were very unsatisfactory. The blame for this can be laid to the radiologists, most of whom are not familiar with the anatomical structures of the sinuses (Grashey's Atlas),[1] nor their pathological lesions. Consequently, the results obtained are unsatisfactory in most instances, and there is great lack of enthusiasm on the part of the radiologist as well as the rhinologist. I am convinced that if a radiologist were to make himself acquainted with the above said conditions, namely, anatomy and pathology of the sinuses, he would obtain just as good results in this line as he does from skiagraphs of stones in the kidney, diseased lungs, etc., and would aid us a great deal in the diagnosis and treatment. Under the present conditions, I believe the best remedy is that the rhinologist assist the radiologist in obtaining the best results. The former is to outline the disease, and the latter does the mechanical part of taking the radiograph and developing it; finally, both study the plate and come to conclusions. That has been my method of procedure.

Albers Schoenberg[2] is one of the few radiologists abroad who has devoted considerable time in developing the radiology of the head, but in this country the credit belongs to Caldwell, who, with the association and co-operation of Coakley, has given us a valuable method of obtaining good plates of the sinuses. The only regret I have is that the method was known to them and not published for such a long time, although presented to a society of radiologists for a year past, that much valuable information could have accumulated by this time on the radiography of the sinuses. I have been and am familiar with a number of rhinologists who also have spent a great deal of time and money on radiographs of the sinuses, only to be disappointed by their results. This I say was

* Read before the Eleventh Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology, Louisville, Ky., September 26-28, 1907.

all due to imperfect technique in not knowing exactly at what angle the rays should traverse the head in order to get a distinct image of the sinuses as to size and pathological condition.

Scheier,[3] in 1897 and 1898, was the first man who proved pus in an antrum by X-ray plate in that he washed out the pus, filled the cavity with water, and showed an entirely different density in the second plate.

Winkler and Brautleicht[4] made a number of observations on X-ray plates taken of skulls devoid of soft parts, but most of their work was done in the transverse view. It was done with especial reference to Winkler's osteoplastic operation on the frontal and ethmoidal sinuses. Mosher[5] has made a number of observations on X-Ray plates, and deduced from them certain measurements of the sinuses. Coakley,[6] in 1905, was about the first to describe and give his results with the use of the X-ray in diagnosis of sinus disease in this country, but so meagre was his report as to the technique that not much knowledge could be obtained from his description. His radiologist, Dr. Caldwell,[7] who, I believe, does the most superior work in this or any other country, read a paper on skiagraphy of the sinuses, but his article did not appear until nearly a year later. Killian and Goodman[8] published their series of experiments on the skiagraphy of sinus diseases only last April, although their work precedes that of Coakley, the latter following their methods. Killian's results are very satisfactory, and the 38 cases that he reports were practically in the pathological condition and anatomical formation which the skiagraphs indicated. Their method in the technique is practically the same that I use and will describe presently. The most recent work published by a rhinologist on the use of the X-ray for sinus diseases is that by Wassermanns,[9] and since his technique, results and conclusions are practically the same as mine, I, therefore, will not go into the details of his article, but present my personal experience and observation, which date back about three years. If I go into greater detail in the technique than necessary for the average radiologist, I hope to be pardoned, as I am speaking principally to rhinologists.

TECHNIQUE.

a. *Equipment*—1. Coil and cord. 2. Table. 3. Compression apparatus. 4. One medium tube. 5. High tube. 6. Plates. 8x10. 7. Developing room with equipment. 8. Transillumination box.

In case stereoscopic views are taken, one needs an apparatus for the examination of such plates, which is a large stereoscope.

b. *Method of Procedure.*—In taking a picture of the sinuses in the antero-posterior direction, the patient is placed on the table face down on to a plate, 8 by 10, in such a manner that the whole top of the head will be reproduced, also the lower margin to include the upper teeth. In that way all the anterior group of cells, frontal ethmoid and antra, on both sides, will be represented. The second step is to adjust the compression apparatus in such a way that when the tube is put in the proper place over the compression apparatus, the rays from the target, which is at a distance of eighteen inches, will pass above the occiput and not through or below it, as was formerly done by many. This is the essential point in the technique that I wish to bring out, for it avoids the dense part of the occipital bone and base of the skull, also in a measure excludes the large sphenoidal sinuses that are liable to mask the other anterior groups of cells, as already mentioned by Coakley in one of his cases.

Caldwell describes a specific method in obtaining the proper angle by taking the base line, which represents the base of the skull, from the external auditory meatus to the glabella, and a line drawn at an angle of twenty-five degrees to this latter line gives the proper angle. The third step is to call attention to the patient, not to start when the current is turned on, but to remain perfectly quiet, although he cannot move his head anyway. It has been the practice of our radiologists to show the patient what the X-rays are, and this has always a quieting effect on neurotic individuals. The current is turned on, 110 volts, 12 amperes, and timed. The length of time we have varied according to the thickness of the skull and the kind of tube we use, whether high, medium or low. As a general rule, with a medium tube, we expose the patient from 60 to 80 seconds, and with a high tube from 30 to 40 seconds, in ordinary thickness of the skull, but in very massive skulls as long as two and a half minutes. The fourth step is to remove the compression apparatus, relieve the patient, take charge of the plate for developing. I might say that for the protection of the plate from perspiration, it is a good idea to place between the plate and the patient an extra layer of waxed paper, else the plates already wrapped in the double envelope may be spoiled.

Fifth, developing the plate. We use Ortol solution, about twenty minutes. The under-development of the plate is a very serious mistake in the technique. Permit to dry thoroughly, from six to eight hours.

Sixth, the examination of the plates, preferably in a darkened room, by means of a transilluminating box. With the aid of the radiologist, we study the outline and changes in the degree of cloudiness or density compared to the normal condition. This is the most interesting part to me, and to compare the results to other methods of examination, as transilluminatin and intranasal inspection.

In taking a skiagraph of a profile, that is, a transverse view, the steps are as follows: First, patient on the side, depending on which side you wish to bring out most clearly. For instance, with a right-sided frontal, ethmoidal and antral or sphenoidal disease, we put our patient on the right side, as the plate will show most clearly the structures nearest to it. Observe the same rule as to the head covering the entire plate. Second step: Compression apparatus. Place so that the rays from the target pass through the base of the skull. This view will also represent a lateral view of the mastoid and middle ear. Third step: Turn on the current and expose again, depending on the thickness of the skull and the degree of the tube. requires a shorter exposure for transverse views, usually from 40 to 60 seconds, with a medium tube. The fourth, fifth and sixth steps are the same as in the antero-posterior exposure.

If one wishes to obtain plates of the antra alone, one can take each side separately for stereoscopic views.

DEMONSTRATION OF NORMAL AND PATHOLOGICAL PLATES OF THE SINUSES.

Plate 1. Shows a skiagraph of a skull taken in the antero-posterior direction. It demonstrates the parts very clearly, owing to the absence of the soft parts. The contrast shows all dense substances white, and spaces dark. The degree of white or black is significant in diagnosis of disease, or absence of the same. The anatomical landmarks are: (1) The orbita, with the superior and inferior margins. (2) The nasal spine, and the lateral wall of the nose, as well as the floor, indicating the nasal fossa, divided by the septum. (3) Malar bones. (4) Superior maxilla, its alveolar processes and teeth. (5) The rami of the lower jaw. The details are: First, the frontal sinuses, with the various subdivisions or septa[10], on the left side, and very interesting fact in this skull, discovered only after the plate was taken, i. e., the absence of the right frontal sinus, only showing the small diploic space, not communicating with the nose in the right side. Second, the ethmoidal cell, with the lacrimal canal in front of it. Third, the antrum of Highmore, in which are seen many white streaks, representing the dense part of bone

of the skull, as the pterygoid plates, etc., but a distinct outline of the wall of this sinus is very clearly shown. You can also observe some other anatomical points not seen when the soft parts are present. (a) Sphenoidal fissure in the orbita; (b) infraorbital foramen; (c) lines indicating the course of the blood channel; (d) spheno-maxillary fossa; (e) the two brass springs are shown most clearly; (f) the black line indicating the horizontal cut of the calvarium usually found in the preparation of a skull.

Plate 2. Same skull taken in the transverse meridian. With the right side next to the plate. It shows the same structures from the side, but not so clearly, however, inasmuch as they are superimposed one on the other. In addition, one can make out the sphenoidal and posterior ethmoidal sinuses, and depths of the antrum.

You will observe the absence of the frontal sinus, which is on the right side. There is no frontal sinus on that side in this skull. Again, you observe the dense base of the skull, with the temporal one. Here the mastoid process with the middle ear cavity is clearly demonstrated.

Plate 3. A normal head, taken in an antero-posterior direction. This man has never had any nasal obstruction, not even a coryza. He has, in fact, never been ill, and is a perfectly developed man, and has as nearly normal nose, throat and ears as one can find. You observe the same anatomical conditions as in the skull, except not so clearly defined, on account of the soft parts. It, however, represents more nearly the appearance of the structures in the study of disease. You observe the uniformity of the shadow of the cavity. The most important point in diagnosing obstruction or pathological changes of the sinuses from normal or thickening of the bone.

Plate 4. Same head, taken in a transverse direction. Same conditions prevail as in Plate 2, of the skull, except the frontal sinus is clearly demonstrated. It shows its antero-posterior diameter as well as its height, but you can see the superimposition of the two frontal sinuses.

Plate 5. A case of double subacute sinusitis involving all the sinuses, following an influenza. All the usual clinical phenomena are present, as pus from the various regions of the sinuses, tenderness on pressure, and the subjective symptoms, as morning periodical headaches and marked mental depression. Transillumination is very unsatisfactory in this case, as both sides are involved. However, the light reflex can be made out in the papillary area. The patient cannot bear the pressure over the frontal sinuses caused by

the transillumination apparatus. The essential point in this plate is the cloudiness of all the anterior group of cells. The patient has been under treatment only three weeks; local irrigation in the region of the ostia with normal salt solution has been used, and he is improving very rapidly. The showing of the plate after recovery is necessary to complete a complementary picture of this plate, and will follow before publication of this paper.

Plate 6. Double chronic frontal sinusitis of right ethmoidal, antral and sphenoidal sinuses, and all the clinical phenomena, as severe headache, especially over the left side, are present. There is pus from the regions of the ostia, and transillumination is positive. Patient has been treated for some time locally by intranasal operations, such as middle turbinectomy and ethmoidal curettement, with but partial relief. Waiting over six months, and following the taking of this plate, we decided to do an external operation. I performed Winkler's osteoplastic operation on the right frontal and ethmoidal sinuses, breaking through the septum into the left frontal sinus, and communicating it with the right side. The Caldwell-Luc antrum operation was performed at a subsequent time, using the Vail[11] saw to make the operation into the lateral wall of the nose. We found during the operation the anatomical and pathological condition exactly as indicated in the plate. Pathologically, the sinus contained some muco-pus and much degenerated muco-periosteum. At another subsequent time I removed the anterior wall of the sphenoidal sinus. For the past six weeks the patient has been well.

Plate 7. Double frontal and ethmoidal sinusitis of a chronic type, with multiple nasal polypi on both sides. No pus. Very severe frontal headache. Intranasal operation of removal of the polypi and middle turbinectomy, curettement of the ethmoid region, breaking down of the anterior wall of the sphenoid, was followed with but partial relief of the severe headaches. Six months later, I performed the Halle procedure[12] into the frontal sinus. This is a very difficult operation and was followed by marked reaction, as a temperature of 104°, very rapid pulse, and it took three weeks before these symptoms subsided and the patient recovered. But at the present time he is very well, and the nose appears to be in good condition.

Plate 8. Multiple nasal polypi, causing complete nasal obstruction for several years. Has asthma and chronic bronchitis; marked headache over the frontal and occipital region constantly. Removed

all the polypi and middle turbinated bodies. Ethmoidal curettement. Opening of the sphenoid. Relief from the asthma and headache for a period of a few months; then the headaches recurring, in fact, increasing, so that an external operation was advised, but refused. You observe a cloudy appearance of these sinuses, that is, the ethmoid and frontal, which shows that the cure is but partial.

Plate 9. Bilateral frontal sinusitis chronica. This man has been suffering from frontal headache for the past two years, almost incessantly, and uncontrollable by any method of treatment. Transillumination negative. Nasal examination: Marked deviated septum and thickening of the middle portion of the same. Sinuses show cloudy. There is no pus discharge. I did a submucous operation, as preliminary to the middle turbinectomy work. Patient very much improved since that operation. A radiograph six months later will be of inestimable value if during all that time he remains free from his headache, which then ought to show clear sinuses.

Plate 10. Pansinusitis chronica, following a severe influenza complicated by acute pansinusitis. The symptoms are extreme dullness of intellect and pain in the head constantly, especially over the frontal region. Pus exuded from region of the sinuses; transillumination dull; antrum and frontal. This skiagraph shows the cloudiness of all the cavities. The usual intranasal treatment was of no avail. Intranasal surgery, as a middle turbinectomy and curettement, breaking-down of the anterior sphenoidal wall, was followed by some relief. Three months later an external operation was advised and performed, and an osteoplastic on the frontal and ethmoidal sinuses. I used in this case Halle drills from above downward, also making a large opening into both antra by means of the Halle angular drills. These procedures were followed by absolute cure. The nose is in as normal condition as one may expect after such procedures. At the time of the operation we found the condition of the sinuses corresponded exactly to the outline in the plate.

Plate 11. Pansinusitis chronica. Having lasted four years, without any relief from treatment or intranasal operations. He has had several attacks of acute frontal sinusitis, with marked pain and some swelling externally, which, however, always subsided in a few days. Transillumination positive of the antrum, but very unsatisfactory of the frontal. External operation; Winkler's osteoplastic, etc., as in case reported in *Plate 10.* Recovery.

Plate 12. Chronic pansinusitis. Local treatment did not cure suppuration. Decided to operate externally. Before so doing, we

took this plate, and it showed that there were no frontal sinuses present, except two small dilatations at the root of the nose. But it does show a very cloudy ethmoid and antrum on each side. Punctures of the antra followed by washing were always associated with pus. We therefore did an intranasal operation, as moderately enlarging the trocar punctures, with the antrum punch, curettage of the ethmoids; also enlargement of the sphenoidal sinus openings. These procedures were followed with good success. The interesting points in this plate are the absence of the frontal sinuses in both views, and it demonstrates the value of this method of diagnosis, for any procedure, whether intranasally or externally on the frontal sinus would have very likely led to serious results.*

DEDUCTIONS IN GENERAL.

1. That the real value of a skiagraph for diagnosis of sinus disease is in taking an antero-posterior exposure, as shown in Plate 1.

2. That the skiagraphs taken in the transverse view are of but very little value for diagnostic purposes, owing to the fact that one side is superimposed upon the other, but it will give an outline of the sphenoidal as well as the anterior group of cells as to their shape and size.

3. That the angle at which the tube is placed is of the greatest value, taking particular care that the rays do not have to penetrate through the massive part of the base of the skull. A transverse line seen across the orbit is found if the angle at which the plate was taken is correct, and this line is found about a half an inch below the supraorbital margin.

4. Dangerous conditions, as burn, alopecia, are possible only if one has no knowledge of the technique, or from carelessness. However, do not expose your patient to repeated long exposures within a brief period of time. I have never had a bad result from the use of the X-rays.

TRANSILLUMINATION.

The value of transillumination of the sinuses must be limited to the frontal and antra, as the ethmoid transillumination is not at all practicable. Although, as said before, skiagraphy will not prove much in posterior ethmoid and sphenoidal disease, it will, however,

* Owing to the unsatisfactory reproduction of the plates for publication, the author decided not to use them, rather than to retouch them for such purpose.

do so in an anterior ethmoidal sinusitis. Transillumination of the frontal sinuses is of very little value, although recently Vohsen[15], the author of this method, defends it in preference to the X-rays, because he says the latter is not practical, and within the reach of very few specialists, as is his little lamp. He lays particular stress on pressing firmly the rubber protection tip of the lamp against the infraorbital margin, and an absolutely dark room. It is quite different in regard to the maxillary antrum. Here the results are fairly satisfactory. The point in the technique of transillumination of the max-

Transillumination Hood.

illary sinus is to have thorough closure of the lips when the lamp is in the mouth. The difficulties are that there is no standard light established. Abroad, the small lamps are used, and in this country the large ones. I am partial to the latter, as my results are more satisfactory with the larger lamps than they were formerly with the small Vienna style.

Another difficulty is in obtaining an entirely dark room. Either it is a stuffy little closet or it requires considerable time to get your office so darkened as to make the procedure practicable. I have, therefore, adopted for the past four years this method of placing this hood over the patient, which is connected with this tube; at its end is the opening which fits very firmly about the eyes, and be-

ing held by this handle with my left hand. With the right is held the transilluminating apparatus, and passed through a slit from below which fits firmly about the wrist. Thus the openings about the examiners eyes and about the patient's head are absolutely lightproof. I have used this method, as said before, for about four years, and am satisfied with it.

N. B. Another plate of No. 5 has been taken since patient recovered (7 weeks later), but the sinuses do not show very much clearer, except that the marginal outlines are sharply defined.

BIBLIOGRAPHY.

1. GRASHEY: Atlas Typischer Röntgenbilder.
2. ALBERS-SHOENBERG: Atlas.
3. SCHEIER: *Arch. f. Laryngol.*, Bd. 6. Heft 1, 1897.
4. WINKLER: Fortschritte und dem gebiete der Röntgenstahlen. Bd. 5, Heft 1, 1901. Bd. 6, 1902-3.
5. COAKLEY: *Ann. of Otol., Rhinol. and Laryngol.*, 1905, Vol. 14.
6. MOSHER: THE LARYNGOSCOPE, 1906.
7. CALDWELL: *American Quarterly ofRoentgenology*, Jan., 1907, Vol. 1, No. 2.
8. KILLIAN and GOODMANS: *Beitr. z. klin. Chir.*, Bd. 54, Heft 1.
9. WASSERMAN: *Internat. Centralbl. f. Ohren.*, Bd. 5, No. 10, July, 1907; and more extensively in Fortschritte auf dem Gebiete der Röntgenstahlen, 1907.
10. HOEVE: THE LARYNGOSCOPE, July, 1907.
11. INGALS.
12. HALLE: THE LARYNGOSCOPE, Feb., 1907.
13. GOOD: *Journ. of Am. Med. Assoc.*, Sept., 1907.
14. VAIL: Transactions of Am. Otol., Rhinol. and Laryngol. Soc., 1907.
15. VOHSEN: *Wien med. Klinik*, No. 23, S. 670.

1220 N. Clarke Street.

THE PREMAXILLARY WINGS AND DEVIATIONS OF THE SEPTUM.*

BY HARRIS PEYTON MOSHER, M.D., BOSTON.

There are two great causes of deviations of the septum, trauma and asymmetry of growth. Of late trauma has been held to play but a minor part. The tendency has been to make unequal development of the halves of the head the chief cause. Undoubtedly this is true in certain cases but more often the inequality of growth is confined to the bones which make the hard palate, namely, the superior maxillae, the palate bones and the premaxillae. I wish to show that irregular and delayed eruption of the teeth, especially of the incisor teeth, is the cause of this asymmetry in a large number of cases. Further, I wish to reinstate trauma as a prominent cause of deviations of the septum.

My attention was called to this subject by a paragraph in Sieur and Jacob's Anatomy of the Nasal Fossae. In the chapter on the septum, in the summary of the causes of deviations, the following statements are found; (page 42); "Hypertrophy of one of the elements making the premaxillary group of bones (the subvomer bone and the nasal spine) appears to play a great part in the formation of crests and spurs which are placed at the entrance to the nasal fossae just behind the nasal spine and appear to be incorporated with the floor. * * * Potiquet has shown that for the subvomer bone in particular the hypertrophy is connected with the eruption of the incisor teeth which are lodged in the premaxillary bones. It seems that this group of bones, originally independent of the superior maxillary bones, undergo to a less extent than these the retrogade changes which all anthropologists have pointed out in the superior races. There can be therefore, not only a failure of parallel growth between the skull and the face but also between the different elements of the face. From this it follows that the projections and ridges of the septum anteriorly which are due to hypertrophy of the subvomer bone or the nasal spine are caused in the last analysis by the same underlying path-

* Read before the Thirteenth Annual Meeting of the American Laryngological, Rhinological and Otological Society, New York City, May 30, 31, and June 1, 1907.

The illustrations were drawn by the writer. From the Anatomical Laboratory of the Harvard Medical School.

ological cause as ridges or spurs which are placed posteriorly, namely—they are due to some disturbance in the development of the face."

I am convinced that this is true. Hypertrophy or displacement of the subvomer bone, however. can do more than cause ridges and spurs just back of the nasal spine. If there is marked delay in the eruption of the incisor teeth not only are the premaxillary wings (I prefer to call the subvomer bones by this name) thrown out of line but the cartilage of the septum is thrown out of its bed in the vomer for a very considerable distance and the septum is deviated in consequence. The starting point of a great many deviations is anterior. The force which causes them is applied anteriorly but is felt not alone anteriorly but posteriorly. At the present time I am not prepared to say just how many deviations of the septum are caused in this way, but since I have been looking into the question I have been surprised to find that so many could be assigned to this cause and I feel that continued clinical work will make the percentage of deviations so caused a high one.

Dentists for a long time have been aware of the connection between crowded teeth and deformities of the septum but as far as I have seen, in a very superficial examination of the literature, the exact relationship between the teeth and the septum has not been brought out. My work has been clinical and anatomical, an endeavor to satisfy myself as to some of the details of this relationship with but little reference to the literature except for the basal idea of the investigation, which as I said is found in Sieur and Jacob and is due to Potiquet.

In discussing my subject, the enlargement and displacement of the premaxillary wings as a cause of spurs and deviations of the septum, I shall do so under the following headings; First, a review of the development and the anatomy of the septum; second, the changes which occur in the premaxillae as the incisor teeth erupt; third, the condition of the septum in forty cases of delayed and irregular dentition; fourth, a description of the examples of deviation of the septum found in the dissection room, where the deviations were due to hypertrophy and displacement of the premaxillary wings; fifth, asymmetry of one-half of the palate as a cause of devations; and finally, trauma as a cause of deviations.

The Nasal Processes. The nasal process at a very early stage in the embryo is seen to be divided into two lateral processes and two

mesial processes, the latter having globular enlargements as tips. These processes are vertical septa springing from the base of the primitive capsule of the fore-brain and the parts seen on the face are the anterior ends of these septa. (Plates 1 and 2.) The mesial nasal processes fuse together and form the whole septum of the nose, the premaxillae and the middle third of the upper lip. They form also the mesial limbs of the alar cartilages.

The Vomer. The vomer is developed from the perichondrium which covers the primitive septal cartilage. A center of ossification appears in the third month at each side of the cartilages posteriorly. These fuse together below. Thus the vomer is at first

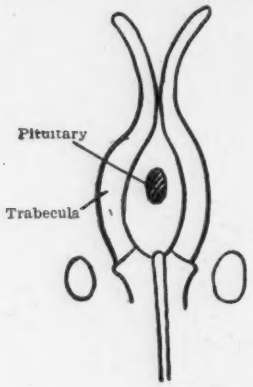


Fig. 1.

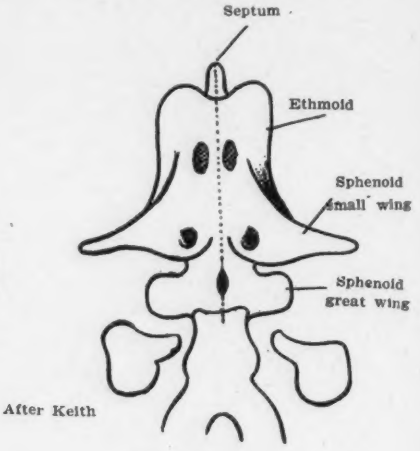


Fig. 2.

PLATE 1.

Figure 1. Diagram of the trabeculae cranii.
Figure 2. The trabeculae have widened and developed into the sphenoid and the ethmoid and septum.

a trough into which the cartilage of the septum is implanted.

The Vertical Plate of the Ethmoid. The vertical plate of the ethmoid is formed by a direct ossification of the primitive cartilage of the seutpm. Ossification begins in the fourth month of foetal life. The crista galli, the intra-cranial part of the septum, is formed in part by ossification proceeding from the attachment of the falx cerebri.

The Premaxillae. The two premaxillary bones form the sockets for the upper incisor teeth. In the human foetus at birth the suture between the maxilla and the premaxilla can be seen on the hard

palate. (Plate 3.) It runs diagonally forward and outward from the anterior palatine canal to the alveolus between the lateral incisor and the canine tooth. On the facial aspect the premaxillae fuse with the superior maxillae in the third month of foetal life, the maxillae overlapping and almost completely excluding them from the face. In mammals generally the premaxillae are highly developed and form the snout. In the higher primates the face becomes less elongated and the premaxillae less developed. In the orang, for instance, the premaxillae are distinctly seen on the face at birth

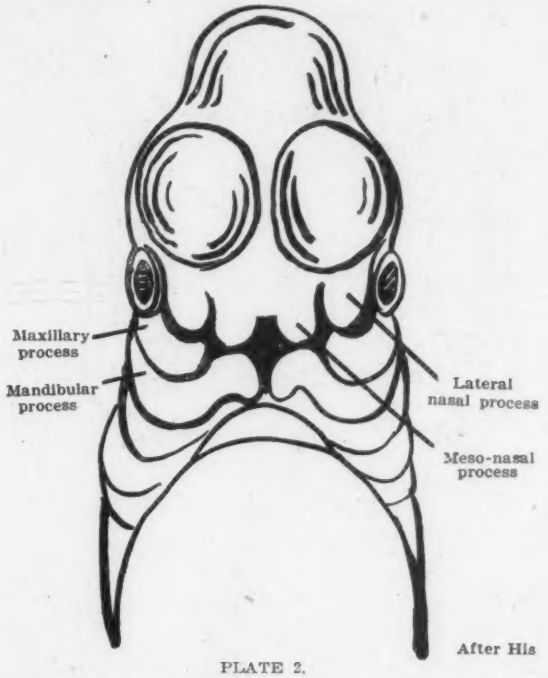


PLATE 2.

Embryo of fourth week showing formation of the face by the nasal, maxillary and mandibular processes.

but as the permanent canines begin to be developed they fuse with the maxillae.

In man each premaxillae is usually ossified from two centers placed side by side. The two premaxillae unite in the first year after birth. Their vestigal character in man is due to the small size of his masticatory apparatus and the consequent retrogression in the development of the facial part of his skull. (Keith, pp. 3-5.)

The result of all this is that the septum becomes ossified from behind forward. Not the whole of it, however, for the anterior part remains as cartilage. This is known as the quadrangular cartilage. I wish to lay especial stress upon two points in the development of the septum. The paired origin of all parts of the septum; and the fact that developmentally the premaxillae are as much parts of the septum as the vomer.

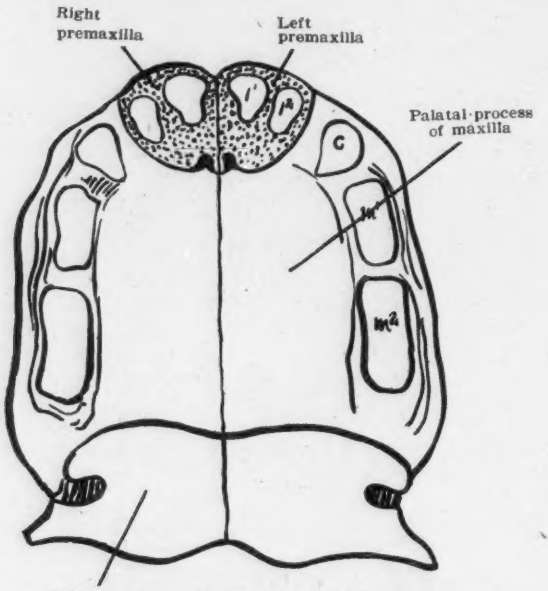


PLATE 3.

The hard palate at birth. The phemaxillary part is formed from the mesial nasal processes. The remainder by the palatial processes of the superior maxilla and of the palate bone.

The Septum at Birth. The septum at birth is almost all cartilage. (Plate 4.) The only bony parts are the vomer and the two premaxillae and their processes. The vomer has a very characteristic form. It consists of two leaves of thin bone united below and open and flaring above. (Plate 5.) This formation is a relic of its double origin, evidences of which the vomer never entirely loses. The premaxillary wings make the vomer over again in miniature. Therefore, they also form a V, or gutter. The premaxillary wings spring from the posterior half of the upper surface of the premaxil-

lae. In the groove which they form rests the tip of the vomer. Two other processes spring from the superior surface of the premaxillae, one from each, namely the nasal spines. These again make a slight gutter, into which in its turn fits the tip of the premaxillary wings. The tip of the vomer rests in the gutter of the

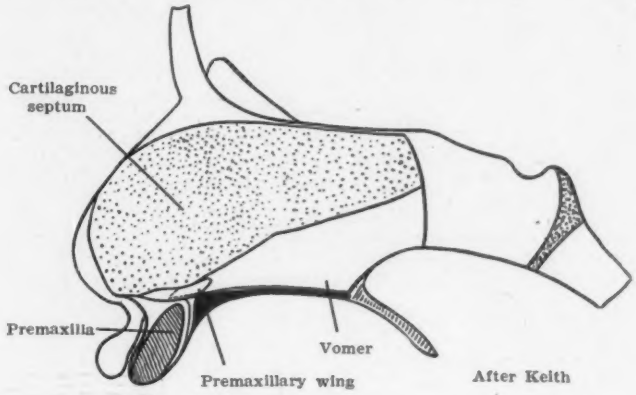


PLATE 4.
The septum at birth.

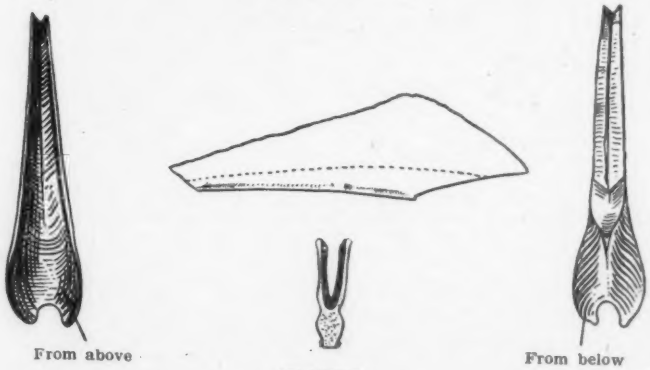


PLATE 5.
The vomer at birth, showing the two leaves and the gutter made by them.

premaxillary wings, and the tip of the premaxillary wings rests in the gutter of nasal spines, like the sections of the old fashioned V-shaped wooden drain. (Plate 6.)

The Adult Septum. The upper border of the adult vomer is gutter shaped like the vomer at birth, the gutter not being so

deep. (Plate 7.) It is surprising how long the vomer retains this characteristic to a very marked degree. At four years of age the vomer still consists of two leaves for half its width. In the adult septum the lower border of the perpendicular plate of the ethmoid is gutter shaped the same as the upper border of the vomer, with which it articulates. Between these two bones, therefore, there is a lozenge like space filled with cartilage. The strip of cartilage which fills this space is an offshoot from the posterior inferior angle of the quadrangular cartilage. It runs backward and upward in the space thus left for it, at times reaching the front wall of the sphenoid. This prolongation is called the caudal prolongation. The following

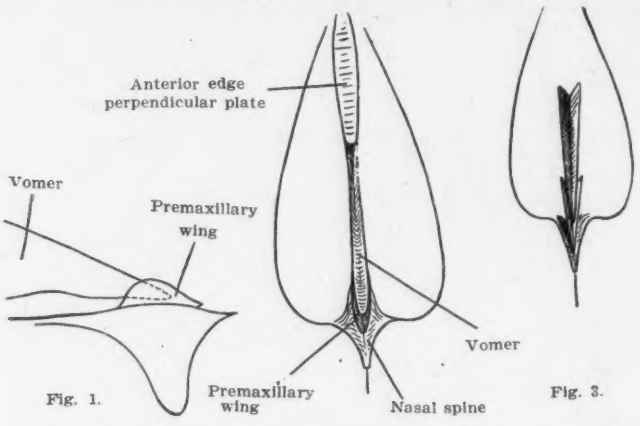


Fig. 1. Fig. 3.

Fig. 2.

PLATE 6.

Fig. 3. is a diagramatic drawing showing how the vomer lies in the premaxillary wings, and the premaxillary wings lie in the gutter of the nasal spine.

are the other peculiarities of the adult septum which have a bearing upon the subject of this paper. The lower part of the quadrangular cartilage just above the premaxillary wings is very thin, whereas the upper part of this same cartilage in line with the middle turbinate is thickened into what is called the tubercle. After eight years of age the further growth of the quadrangular cartilage occurs chiefly at a center of growth just above the premaxillary wings. There is a second less important center of growth half way up its posterior border. At birth neither the palate bones nor the superior maxillae rise into a crest for the support of the lower edge of the septum. (See Plate 4.) In the adult on the other hand both

these bones have marked crests. These grow upwards while the rest of the septum is growing downward affording at times, another factor in disturbing the equilibrium of the mosaic of the septum. This factor is not generally considered.

It has usually been held that the weakest part of the septum is the articulation between the upper border of the vomer and the lower edge of the perpendicular plate of the ethmoid. From the description of the septum which has just been given it is easy

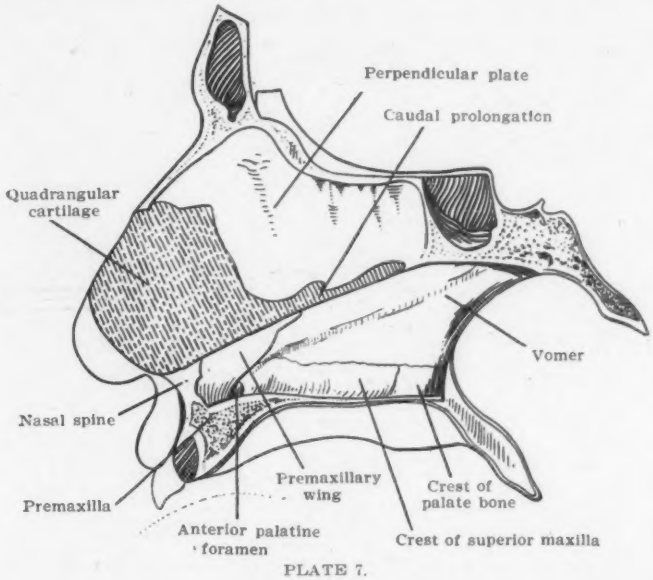


PLATE 7.

The adult septum.

to see why this is so. There is, however, another part of the septum which is as weak or weaker than this suture line. This place is at the tip of the vomer, or the region of the premaxillary wings. Here the mosaic of the septum is the smallest, here the tip of the vomer lies in the groove of the premaxillary wings, here the caudal prolongation of the quadrangular cartilage starts, here the quadrangular cartilage is the thinnest, here is placed the remnant of the organ of Jacobson, and here the quadrangular cartilage has its chief center of growth. Anatomically, therefore, this part of the septum is especially fitted to be the starting point of spurs and

deviations. If for any reason the equilibrium of the septum is upset one would expect it to show itself first at this point.

The two premaxillae do not fuse until well into the first year. When they are disarticulated each premaxilla is seen to be an oval shell of bone, all the faces of which are very thin. The body of the bone is completely filled with the crowns of the incisor teeth and accurately takes its form from them. (Plate 8.) Projecting

Fig. 1. Side view.

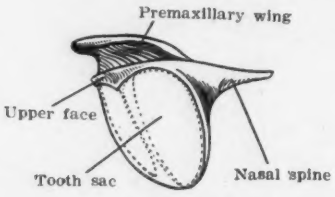


Fig. 2. Front view.

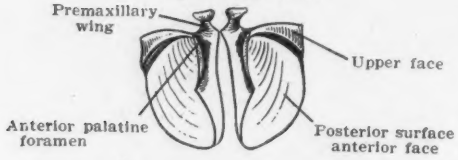


Fig 3. Premaxilla seen from above.

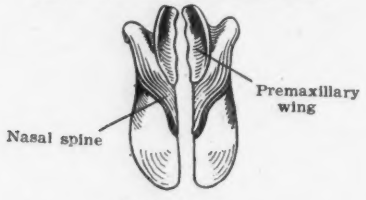


PLATE 8.

The premaxilla at birth.

horizontally forward from the anterior edge of the upper face of the premaxilla is a pointed process called the nasal spine. The superior surface of this is slightly concave. Also projecting from the upper face of the premaxilla in the middle line, but projecting obliquely upward and outward, is another process, the premaxillary wing. This process extends along the posterior two-thirds of the superior face of the premaxilla. Its upper surface is markedly concave. The inner edge of the base of this process is irregularly undermined so

that it can mortice into the corresponding process of the other premaxilla. The upper edge of the wing slopes outward and the root of the wing on its outer side is constricted by a smooth gutter, as if for a vessel or a nerve. Owing to this the wing has a narrow constricted base and a concave flaring superior surface. When the two premaxillary wings are placed together the wings mortice into one another so that they form a sizable gutter. Into this, in the complete septum, fits the tip of the vomer also a gutter. The premaxillary wings act as cleats on either side of the tip of the vomer to hold it in place. Their position controls the position of the vomer.

From birth up to six years of age the premaxillary wings increase in size but little. From this time on, however, they en-

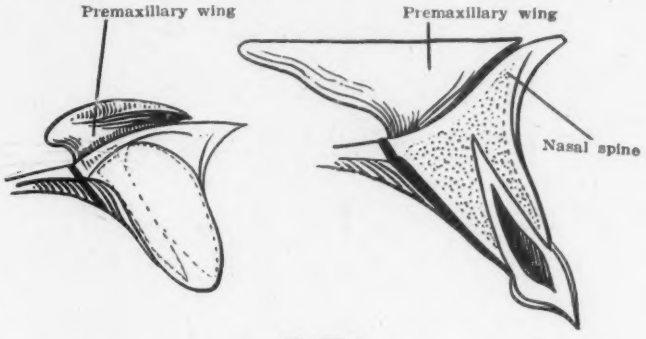


PLATE 9.

Fig. 1. Premaxilla at birth. Fig. 2. The premaxilla in the adult.

large rapidly, so that in the adult each wing measures about half an inch along its upper border and is a quarter of an inch in height. (Plate 9.) The premaxillary wing grows especially upward and backward so that its final form is triangular. The apex of the triangular piece of bone which constitutes the premaxillary wing points downward and rests just in front of the anterior palatine canal. These processes undergo a greater proportional growth than almost any other part of the septum. This is necessary in order that the premaxillary wings should not lose control of the vomer.

The premaxillary wings are said to fuse with the tip of the vomer at fifteen years of age. On account of their position and on acount of this fusion with the vomer the two processes have usually been known as the sub vomer bones. Dissection has led

me to consider them not as separate bones but as processes of the premaxillae the same as the nasal spines. At birth the premaxillary wing is firmly united to the superior face of the premaxilla by a slender neck and apparently is a part of it. For this reason I call these processes the premaxillary wings.

As the antra enlarge and descend with the eruption of the second teeth the crest of the palate bone and the crest of the superior

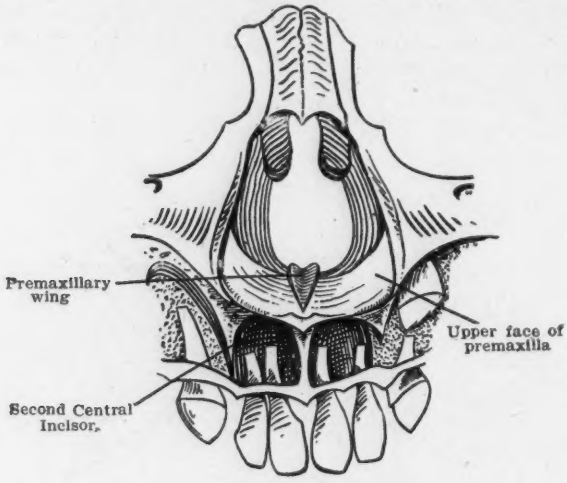


Fig. 1.

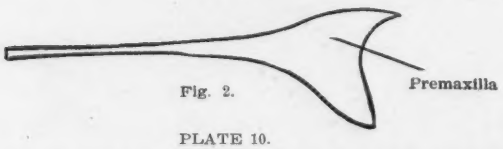


Fig. 2.

PLATE 10.

Fig. 1. Dissected alveolus of a child three years old. The upper face of the premaxilla is flat and fits accurately over the tops of the second incisor.
Fig. 2. Shows that the upper face of the premaxilla is almost flat.

maxilla grow upward. (Plate 10.) At the same time the nasal spines and the premaxillary wings also grow upward. Heretofore the superior face of the premaxilla has been flat like a plateau. This plateau disappears and the superior surface slopes downward and backward at an angle of forty-five degrees. The upward growth of the nasal spine is in great measure responsible for this. The spine in the adult makes a bony rim for the vestibule and turns the incisor plateau of infancy into a moat.

The fact that the inclination of the superior face of the premaxilla depends upon the teeth below it and changes as these erupt is shown prettily by dissections. At birth the upper rim of the hollow crown of the middle incisor fits snugly under the thin

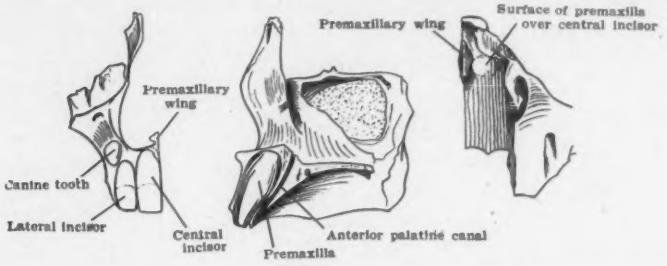


Fig. 1. Fig. 2. Fig. 3.

PLATE 11.

The premaxilla and the superior maxilla from a child of six.

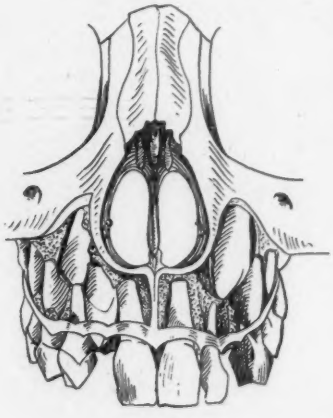


Fig. 1.

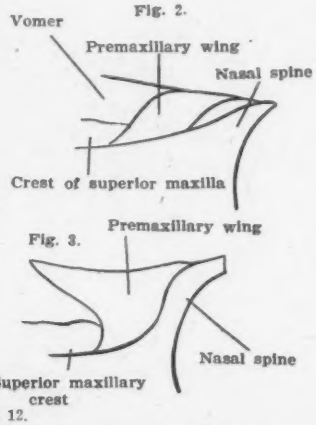


PLATE 12.

Fig. 1. Dissected alveolus of a child eight years. The nasal notch has lost its former triangular shape.

Fig. 2. The upper face of the premaxilla. The nasal spine and the premaxillary wing are beginning to grow upward.

Fig. 3. The nasal spine and the premaxilla have their adult growth.

upper face of the premaxilla. The upper rim of the crown is flat and so is the superior face of the premaxilla which is paper like in thickness and roofs it over. Plate 10, which is drawn from

the dissected alveolus of a three year old child shows this. As the flat rim of the upper part of the hollow crown gives place to the pointed root the superior face of the premaxilla changes from a flat plateau to a slanting surface. The form of the nasal notch changes in the meantime from a triangular aperture to a heart-shaped opening. Normally, therefore, the line of the superior

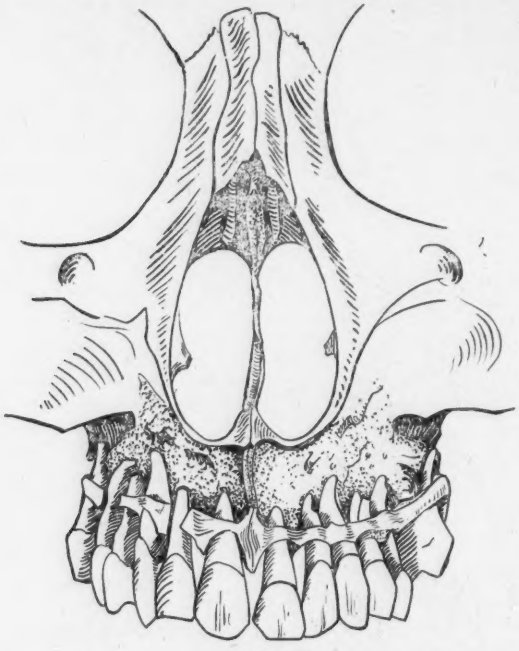


PLATE 13.

Dissected alveolus in adult. The nasal notches have the adult heart-shaped form. (See PLATE 12, Fig. 3.)

face of the premaxilla and the shape of the nasal notch are determined by the degree of eruption of the incisor teeth. Plate 11 represents the superior maxilla and the corresponding premaxilla at six years. In this specimen the upper rim of the crown of the central incisor is flush with the under surface of the superior face of the premaxilla and occupies the greater part of it. The layer of bone over the tooth crown is still paper thin, whereas the amount of bone over the lateral incisor is very thick. The lateral incisor could have but little influence as compared with the

central incisor upon the form of the superior face of the premaxilla and upon the premaxillary wing. (Plate 12.) All other parts of the alveolus except the part made by the premaxillae are buttressed by the walls of the antrum. (Plate 13.) The superior face of the premaxilla is free in the nose and not so buttressed. The form of this, therefore, can be readily altered.

Of late the old theory that the eruption of the teeth is caused by the formation of the root and pressure upon this as it elongates has been somewhat questioned. The new theory is that the eruption of a tooth is due to the increase of the blood pressure within the tooth sac. The tooth is forced out, so to speak, by hydraulic pressure. This may be true in part but it is not the whole truth. The anatomical facts which have just been given and the clinical findings which are to be given next in order show that the presence, or better the pressure, caused by the tooth root determines the form of the superior face of the premaxilla.

Th blood pressure theory is of interest for this reason. Hutchinson's teeth is the name of a familiar deformity of the incisor teeth, especially of the central incisors. Inherited syphilis, which causes the deformity, involves in the nose chiefly the septum. Developmentally and practically, therefore, the premaxillae are a part of the septum. The incisor teeth are likewise in a way a part of the septum; and, in common with it, suffer from the syphilitic invasion. Thus it comes about that they are selected over all the other teeth for excessive malformation. The fundamental pathological change in syphilis is an endarteritis. If blood pressure has a part in causing the eruption of the teeth the pressure would be interfered with by the arterial changes. To interfere with the permeability of an artery is to interfere with the nutrition of the part which it supplies. Therefore we generally find that all the teeth in cases of inherited syphilis are stunted.

If the normal eruption of the incisor teeth causes a change in the upper face of the premaxilla the question is at once suggested: Does delayed or irregular eruption of the incisor teeth cause a still further change, a change which is of any significance in the pathology of the septum? It does. Within the last month I have collected forty cases of irregular and delayed eruption of the incisor teeth. The patients were mostly children who came to the clinic to have their tonsils and adenoids removed. The ages of the majority were between seven years and twelve. Five of the cases were adults. Through the kindness of Dr. L. B. LeGro, of Haver-

hill, I had the opportunity to examine four cases from his private practice where the history of the teeth was known from the beginning and where the teeth had been carefully followed and cared for. Since every point in connection with these cases could be followed I will give the cases in detail.

T. B., female, 8 *years.* Eruption of the incisors six months ahead of time. Right middle incisor fully down and in good line. Left middle incisor just appearing through the gum. Both first lateral incisors lost, neither second lateral incisor showing.

Nasal examination. The arch of the palate is moderately high Both halves of the arch of the palate are of equal height. The wing of the premaxilla on the side of the left central incisor, the delayed incisor, is enlarged. There is a left vomer spur running back from this for one-half inch. The right premaxillary wing is not enlarged. The septum deviates moderately to the right, for three quarters of an inch back.

There is no nasal obstruction. Tonsils and adenoid were removed three years ago. No tonsils are to be seen at the examination.

G., male, 8½ *years.* Both central incisors fully erupted and in perfect line, at the present time, but the patient's history card states that the left middle incisor came down six months after the right middle incisor and six months after the normal time of eruption. The arch of the palate is normal and both halves are of equal height.

Nasal examination. There is a slight vomer spur on the left, and a slight deviation of the septum to the right beginning about a third the way up on the septum and running one-half inch back. There is a slight vomer spur on the right. No nasal obstruction. Tonsils and adenoid were removed three years ago. Practically no tonsils are to be seen.

M., female, 18 *years.* The left incisors and the left canine were delayed in eruption, and were irregular and crowded. Much regulating has been done to the teeth and the first bicuspid has been removed in order to allow the teeth in front to come into line.

Nasal examination. The premaxillary wing on the left is slightly enlarged with a slight deviation of the septum into the right side. The axis of the deviation is horizontal. There is moderate enlargement of the wing of the premaxilla on the right running back one-half inch. Half way back on the vomer-ethmoid junction there is a small sharp spur. The arch of the palate is normal. Both halves are of equal height. There is slight chronic rhinitis, and a moderate granular pharyngitis.

M., male, 9 years. All the upper incisors of the first dentition are still in place. Normally these should have erupted two years ago. The incisor teeth are regular in outline but are movable on pressure as if they were on the point of being cast off. The nasal bones are in good line. There is no history of trauma. There is no nasal obstruction. The arch of the palate is normal and both halves are of equal height.

Nasal examination. Large vomer spur is on the left, running well back. The left premaxillary wing is much enlarged, the right premaxillary wing slightly enlarged. The septum deviate into the right nostril. The axis of the deviation is horizontal.

These four cases are from families in good circumstances so that the teeth have had every chance. In all, the line of the nasal bones was straight. In all, careful questioning of the parents brought out no history of trauma. In the first three cases there was moderate delay and irregularity in the eruption of the incisor teeth. In these examination showed an enlargement of the premaxillary wing on the side of the delayed tooth and a slight deviation of the anterior part of the septum. In the fourth case the first teeth were retained two years beyond the time when they should be shed normally. In this case there was a very large premaxillary wing on one side with a large vomer spur running well back from it, a moderate enlargement of the other. and in addition a deviation of the septum into the opposite side. So marked was the deviation that any increase from continued growth along the present vicious lines or any sudden increase from trauma must produce obstruction.

The case of the girl of eighteen is interesting because she started with delayed and crowded teeth. Her teeth were carefully regulated, however, and brought into line so that her septum escaped with but slight deformity. The lesson of such a case is obvious. At the time that I saw the second of these four cases both central incisors were equally and fully erupted and in good line but the history card showed that the left middle incisor came down six months after the right middle incisor and six months delayed. In the nose, on the side of the lagging incisor, examination showed that the premaxillary wing was enlarged and that there was a small vomer spur on the same side. The septum deviated slightly to the right. From these cases it is seen that the history of the teeth is to be read in the nose.

The results of the findings in the rest of the forty cases can be put very briefly. They are as follows: Wherever there was moderate and equal delay in the eruption of the central incisors the premaxillary wings were symmetrically enlarged at the floor of the nose on both sides. In such cases there was usually little if any deviation of the septum and there was no vomer spur. Where, however, there was marked inequality and delay in the eruption of one central incisor as compared with the other central incisor, on the side of the backward tooth the premaxillary wing was much enlarged or displaced and the quadrangular cartilage tipped out of its bed along the vomer ethmoid suture. As a rule the long axis of the deviation was antero-posterior, roughly paralleling the spur,

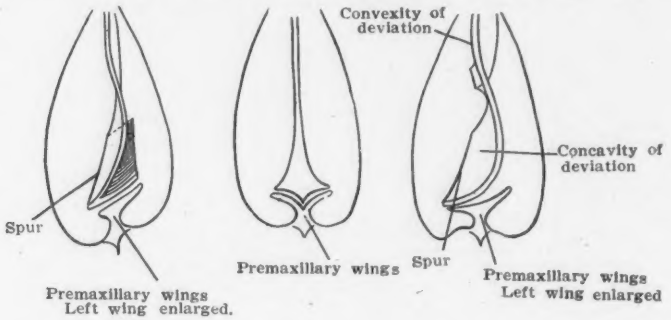


Fig. 1. Fig. 2. Fig. 3.

PLATE 14.

Diagramatic drawings of the types of deviation due to deformity of the premaxillary wings.

Fig. 2. Both premaxillary wings turned outward, causing a double basal spur of the septum.

and the upper part of the cartilage was bowed toward the spur. Accompanying this horizontal deviation there was often a certain amount of sigmoid deviation, the axis of which was vertical. It is fairly common to find the root of the lateral incisor mounting into the outer part of the floor of the nose. Even when the lateral incisors are crowded out of the dental curve and are placed directly behind the central incisors, a deformity which is striking and one from which you would expect much at first glance, they seem to have but little influence on the septum as compared with the central incisors.

The findings of these forty clinical cases are borne out and the details of their formation explained, by the dissection of twelve

cases in which an enlarged or displaced premaxillary wing caused a deviation of the septum. These specimens showed that if the pressure caused by the premaxillary wing is applied to the septum directly in its vertical axis, the lower edge of the cartilage stays in its bed but that the lower part of it folds sharply upon itself, the axis of the fold paralleling the upper border of the vomer. (Plate 14.) More often, however, the force seems to be applied a little to one side of the vertical axis of the septum. When this happens, the lower rim of the cartilage is forced out of its groove be-

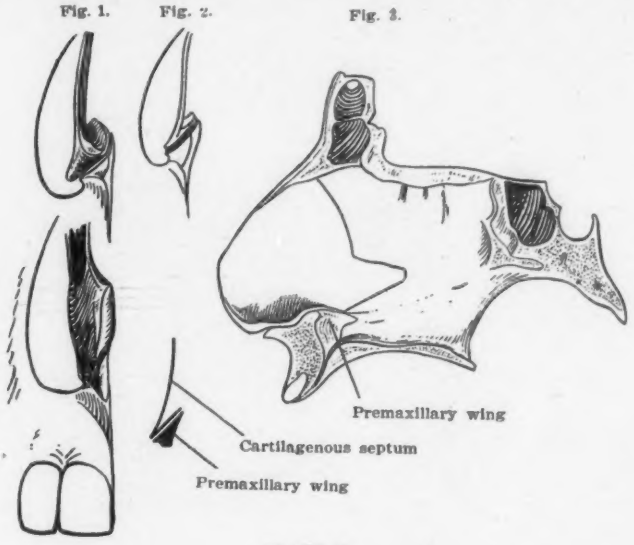


PLATE 15.

Specimen of deviation of the septum due to enlarged left premaxillary wing. Fig. 1. Dissection of the groove of the premaxillary wing. Left wing enlarged. Fig. 2. Scheme of the deviation.

tween the retaining premaxillary wings and the two leaves of the vomer and curls upward and outward. As it does this it breaks off one premaxillary wing or stunts its growth and does the same thing to one leaf of the vomer groove. This results in the familiar spur along the vomer ethmoid articulation. (Plate 15.) The concavity of the lower portion of the cartilage about half way up the septum merges into a compensatory convexity. These dissected specimens bear out the clinical findings that the convexity generally is toward the spur and away from the enlarged premaxillary wing. (Plate 16.)

Occasionally the reverse of this is found. The spur is on the side of the convexity and seems to crown it. The enlarged premaxillary wing is found in the dissected specimen to make an anterior basal spur which runs for a quarter of an inch to an inch backward. Further, when the two premaxillary wings are enlarged but slightly and are enlarged equally they often tip outward and make a small anterior basal spur of either side of the septum. Where, however, both premaxillary wings are evenly enlarged to a considerable extent and the pressure caused by this enlargement is not equalized by

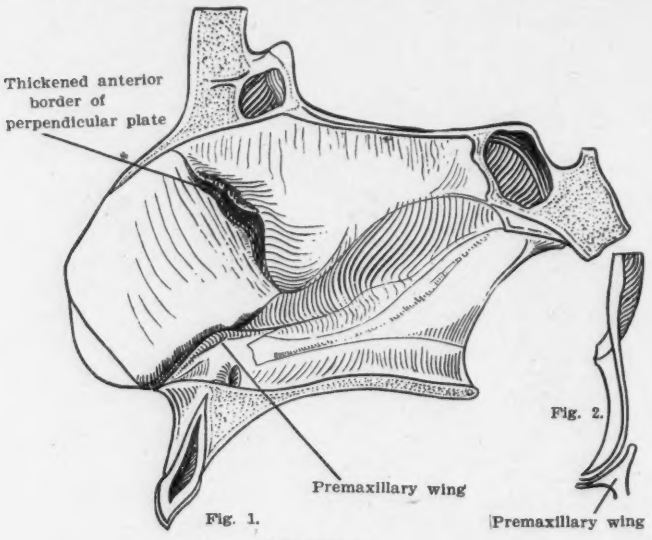


PLATE 16.

Fig. 1. Deviation due to enlargement of the premaxillary wing.
Fig. 2. Dissection of the deviation.

their turning outward, as in the case just described, the tip of the septum is first pushed upward and then to one side. (Plate 17.) The septum in this case yields instead of the premaxillary wings. I found this condition in the skull of a three year old child.

The spur, as has just been said, usually is on the side of the convexity and often seems to crown it. The character of the spur naturally varies with its formation. Where the spur is made by a simple folding of the lower part of the cartilage upon itself the spur is all cartilage. On the other hand, where the cartilage breaks down one leaf of the vomer the spur would be cartilage above and

bone below. Where the vomer groove is distorted but not broken the spur would have a shell of bone above and below with a core of cartilage between. If one premaxillary wing is broken off or atrophies from pressure and the same thing happens to the corresponding leaf of the vomer the spur would again be all cartilage. It is common in the clinic to see a vomer spur end about two-thirds the way back on the septum in a sharp spine like projection. A dissection of such a case shows that this formation is due to the fact that the two leaves made by the lower edge of the perpendicular plate of the ethmoid and the two more pronounced leaves of the upper border of the vomer turn half round until they coincide

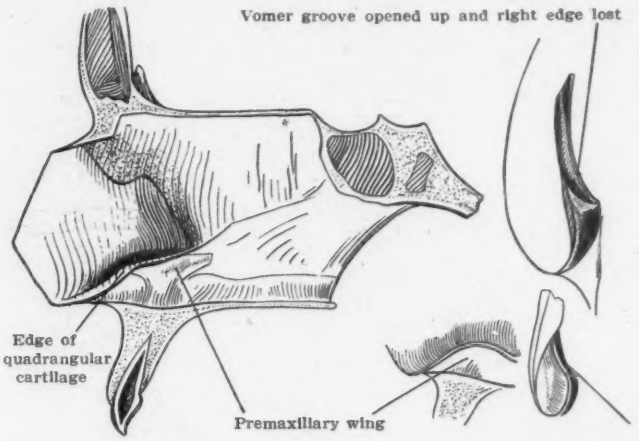


PLATE 17.

Deviation of the septum due to the enlargement of the left premaxillary wing. Small figures, the dissected anatomy of the deviation.

with the long axis of the septum and then bend sharply outward and burst open. (Plate 18.) This spine-like excrescence may be all bone and very hard or it may have a core of cartilage.

Enlargement of the nasal spine, as well as enlargement of the premaxillary wing, can cause spurs and deviations of the septum. Enlargement of the nasal spine does occur but it is less common to find examples of it in the dissecting room than it is to find enlargement of the premaxillary wing. It should be borne in mind however ,that the nasal spine is the cause of deviations in certain cases. In the cleaned skull the nasal notch on the side of the en-

larged premaxillary wing is higher than the notch of the other side. A good X-Ray will occasionally demonstrate this in the living.

I feel that good anatomical and clinical evidence has been given to show that enlargement of the premaxillary wings, the enlargement being due to delayed eruption of the incisor teeth, must be considered as important cause of deviations of the septum. There is yet more to be said about the teeth. Not only may the anterior openings of the nose be of unequal height and size, but also the posterior openings, the choanae. (Plate 19.) The anterior central teeth produce the deformity in front and what might be called the posterior central teeth, the upper wisdom teeth, produce the deform-

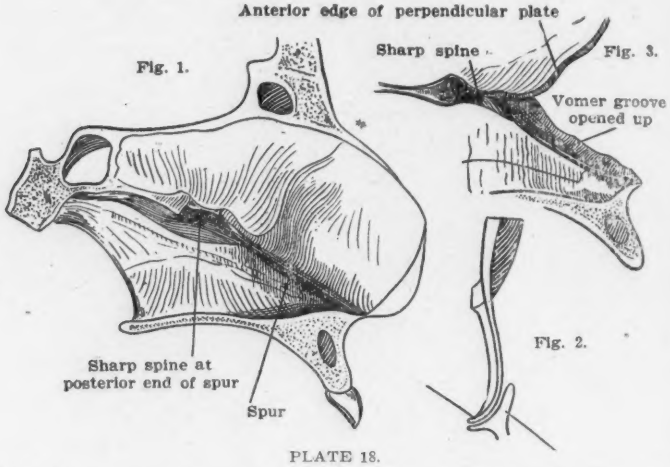


PLATE 18.

Deviation of the septum caused by enlargement of the left premaxillary wing. Fig. 2. Anatomy of the deviation and spur.

ity behind. The molar teeth and especially the wisdom teeth are brought into place by the downward growth of the tuberosity of the superior maxilla in which they are lodged. This shuts into place like a hinged box cover. At times it does not shut completely down leaving the wisdom teeth to erupt at a vicious angle. The palate bone which forms the outer side and the floor of the choana is fastened to the inner side of the tuberosity of the superior maxilla and must follow its movements. If, therefore, the normal downward growth of the tuberosity, and in consequence the normal eruption of the wisdom teeth. is disturbed, this can cause an inequality of the nasal notches. When it occurs it probably plays

a certain part in upsetting the equilibrium of the different parts of the septum.

In the four clinical cases which were given in detail I was careful to state that in all, the two halves of the arch of the palte were of equal height. I did this because there is a third cause of deviations of the septum which if it is connected with the teeth is connected with all the teeth of one side not as in the other cases with special teeth. The third cause is unequal descent of the antra. It results in asymmetry of the whole of one-half of the palate. In such in-

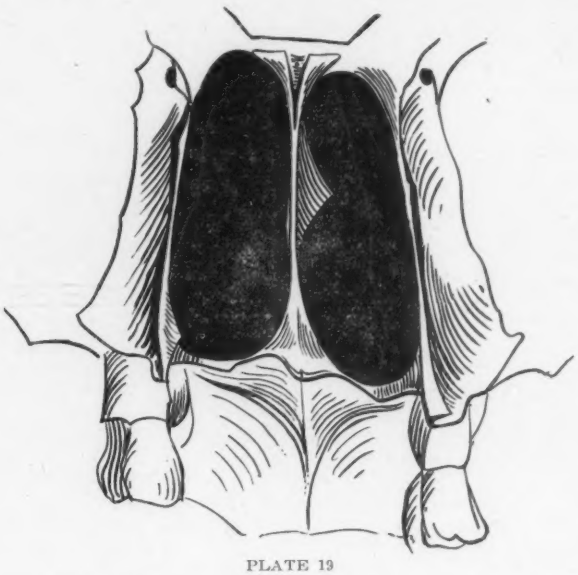


PLATE 19

Inequality of the choanae due to irregular descent of the antra. The left antrum has not descended as low as the right, so that the left choana is higher than the right choana.

stances one-half of the palate is higher than the other half. Plate 20 illustrates this condition . The left nasal notch, the left choana, and the half of the palate between, are higher than the corresponding parts of the other side. This has caused hypertrophy and displacement of the left premaxillary wing and an opening up of the gutter made by them, and the displacement of the tip of the vomer toward the lower nasal notch. This inequality in the halves of the palate is readily seen in the living by looking for it

in the mouth. I found an example of this recently in a child of six years. Small differences in the antra are hard to make out in the X-ray plate, but in one of my adult cases the plate showed clearly that it was present.

When one nasal notch is higher than the other nature often attempts to equalize the deformity. For example, if the right nasal notch is higher than the left just the reverse of this may be true of the choanae. In these cases of inequality of the halves of the

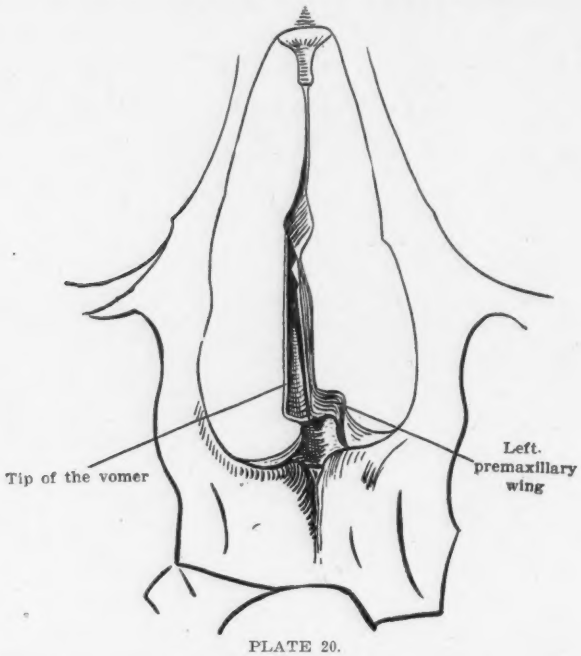


PLATE 20.

Specimen shown in Fig. 19 seen from the front. The left nasal notch is higher than the right. The left premaxillary wing is enlarged and has thrown the tip of the vomer to the right.

palate the asymmetry may extend to the whole half of the head, but as a rule it is confined to the bones which make the hard palate and have to do with the teeth. Where, however, there is asymmetry of half of the head the nasal bones and the septum share in the general asymmetry. In such cases the cartilage of the nose both externally and internally follows the line of the deviated nasal bones.

The last great cause of deviations of the septum is trauma. It used to be argued against trauma that the less civilized races, where there is the most chance for trauma, have straighter septa than the civilized races. This has been in great part disproved by the examination of prehistoric skulls. The negro may have a straighter septum than a white man. If he does it is probably due to the fact that the negro has the heavy premaxilla of the carnivora. The resemblance is very striking. The bony vestibule of the nose of a negro is wide open and flaring and roofed with thick bone, just like, for example, the premaxilla of a bear. Owing to the strength of the premaxilla his septum is kept in good line.

The septum at birth for the greater part is cartilage. It is so resilient that one can study the effect of trauma upon it with ease. For the first year the articulations about the superior maxilla are so soft that it is possible by upward pressure in the mouth to collapse the face to a certain extent. It is somewhat startling to do this for the first time. By putting two fingers of each hand in a baby's mouth under the premaxillae and getting counter pressure upon the skull with the thumbs the face can be shut together very appreciably. This spring of the face must be a great safeguard during birth and early in infancy. I have often wondered how the septum fared at birth. By experimenting with a wet specimen of a baby's septum it is easy to see what must happen. During birth the nose is flattened with the face. In instrumental deliveries the flattening is extreme. In order for this to take place the septum folds upon itself vertically. After birth either the natural spring of the cartilage or the first wiping of the baby's nose straightens this deviation out. An instrumental delivery, however, might readily throw the septum out of its groove in the premaxillary wings or break one of the leaves of the vomer and so make the deviation permanent.

If pressure is applied to the baby's septum from below the cartilage folds horizontally the same as a playing card in the half closed palm. The septum is not ossified to its full extent until the eighth year. Up to this time, therefore, blows upon the septum, unless extreme, are probably taken care of by the spring of the cartilage. Great violence could either jump the tip of the vomer out of its bed in the premaxillary wings or fracture one or both of these wings and one or both of the leaves of the vomer.

After the perpendicular plate of the ethmoid has ossified all that it is to ossify its anterior border plays an important part in

deviations caused by trauma. The under surface of the median border of each nasal bone is supported from above downwards usually for two-thirds of its extent by the superior border of the perpendicular plate of the ethmoid. The anterior border of the perpendicular plate of the ethmoid, therefore, starts from a point a third of the distance from the tip of the nasal bone to its root and runs obliquely downward and backward to meet the upper border

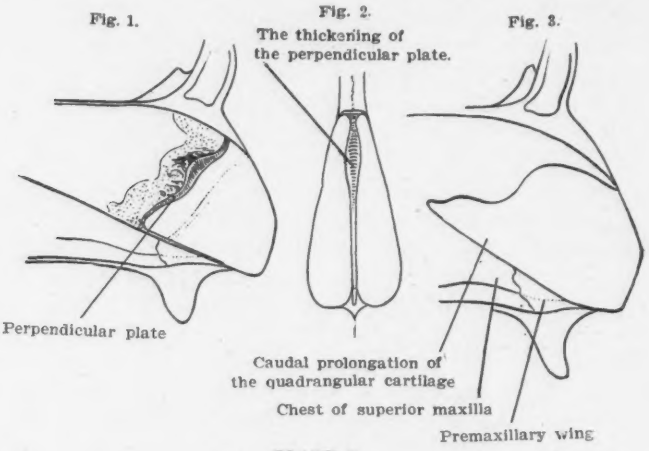


PLATE 21.

Fig. 1. A part of the quadrangular cartilage has been cut away in order to show the thickening of the anterior edge of the perpendicular plate of the ethmoid. This edge is ossified for some distance back.

Fig. 2. The thickening of the perpendicular plate.

Fig. 3. The anterior edge of the perpendicular plate runs horizontally backward and makes a large caudal prolongation.

of the vomer just behind the premaxillary wings. (Plate 21.) The direction of the anterior border of the perpendicular plates of the ethmoid, however, is subjected to great variation. Instead of being roughly vertical as has just been described, it may run backward almost horizontally, in this way increasing the size or the caudal prolongation of the quadrangular cartilage, and turning the anterior inferior angle of the perpendicular plate of the ethmoid into a tongue-like projection.

Where the direction of the anterior border of the perpendicular plate of the ethmoid is practically vertical it is well fitted to withstand trauma. In such a case the force of a blow on the nose directly

from the front is transmitted by the quadrangular cartilage squarely to this edge of the perpendicular plate and most of the force expands itself there. A hard blow produces a vertical deviation or folding of the quadrangular cartilage beginning below at the premaxillary wings and running upward roughly paralleling the anterior border of the perpendicular plate of the ethmoid. Experimental work on the cadaver has proven that the articulation between the anterior border of the perpendicular plate of the ethmoid and the quadrangular cartilage is often too firm to be broken except by the greatest force. herefore the deviation parallels the synchondrosis but does not invole it.

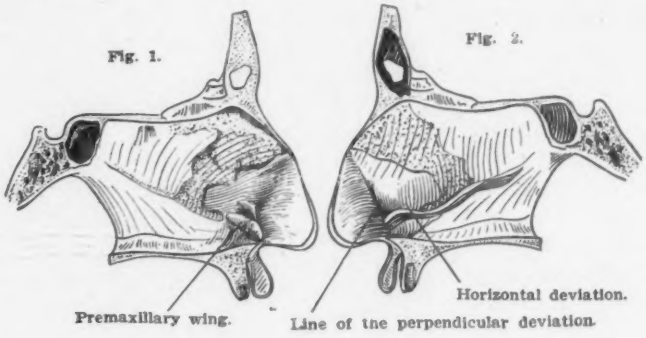


PLATE 22.

Plate showing a perpendicular deviation of the septum with enlargement of the right premaxillary wing. This type of deviation is characteristic of trauma. In this case, both the displacement of the premaxillary wing and the deviation were probably caused by trauma.

Where the anterior border of the perpendicular plate of the ethmoid is vertical, it is adapted to withstand trauma, but where this border runs backward horizontally, it loses its function of a buffer. In this case it is not rightly placed to receive and withstand the effect of a blow from the front. The caudal prolongation directs the force beneath and beyond it, well into the middle of the septum. (Plate 21.) The tip of the vomer is forced from its bed between the premaxillary wings and one or both of these processes are broken. The caudal prolongation is driven as a wedge between the upper border of the vomer and the tongue-like projection of the anterior inferior angle of the perpendicular plate of the ethmoid. The anterior inferior angle of the perpendicular plate bends in consequence and bends or breaks one leaf of the vomer groove. The size of the

caudal prolongation therefore, in great measure determines the form and the extent of the deviation produced by trauma.

The anterior edge of the perpendicular plate of the ethmoid is a natural buffer not only on account of its position but also on account of its thickness. The upper half of this edge is the thickest part of the septum. This thickening occurs on a level with the middle turbinate, and extends backward into the body of the ethmoid plate from a quarter to half an inch. Often this thickening is covered with exostoses. It would seem that these are probably due to the constantly repeated small injuries of childhood.

In short, a sharp anterior vertical deviation is characteristic of trauma. Such a deviation begins below at the premaxillary wings and runs obliquely upward paralleling the anterior edge of the perpendicular plate of the ethmoid. The perpendicular deviation almost always merges into a horizontal deviation which includes the anterior inferior angle of the perpendicular plate of the ethmoid. The shape of this part of the ethmoid plate materially influences the form of the deviation. (Plate 22.) This horizontal deviation generally turns to the same side as the vertical deviation.

Excessive trauma, applied either from the front or from the side, causes deviations and fractures of the septum which cannot be brought into any classification.

Treatment. I have nothing to say about treatment. That is not included in my subject. I wish, however, to add a word concerning prophylaxis. The obvious thing to do is not to leave the first incisors in too long. Dentists as a rule object to early extraction of the first teeth because they say that the second teeth erupt irregularly if the first teeth come out too soon. If this is so, there are two horns to the dilemma, namely, leave the first teeth in as long as possible and deform the septum or remove the first teeth early and deform the second, but keep the septum straight. Most men and all women would prefer straight teeth and a crooked septum to a straight septum and crooked teeth.

Just at present there is a good deal in dental literature about widening the arch of the palate and so widening the nasal fossae. The old fashioned split plate would undoubtedly accomplish this. It is yet to be demonstrated beyond a doubt that the modern methods accomplish more as a rule than a change in the angle of the teeth and an apparent widening of the arch. It is to be hoped that it will soon be proved that the nasal fossae can be widened by widening the arch of the palate. Because, if it can be brought

about to any appreciable extent, the dentist can do much to prevent and to correct deformities of the septum. Dentists maintain that they can ensure by treatment the regular and normal eruption of the incisor teeth. By accomplishing this they can save many a septum from deformity.

Summary.—A large number of deviations of the septum are caused by asymmetry in the development of the bones which make the hard palate. This inequality of development is usually due to delayed or irregular eruption of the incisor teeth, especially of the middle incisor. Delayed eruption of the teeth is caused in great measure by some disturbance of nutrition. When the eruption of one central incisor is sufficiently delayed it causes a deformity or hypertrophy of the premaxillary wing above it. This distorts the retaining groove made by the premaxillary wings. As a result, the septum slips from its bed in the vomer, and the groove made by two leaves of the vomer spreads open, one leaf or side of the V disappearing. This produces a spur along the upper edge of the vomer. As the cartilaginous part of the septum slips from its bed, the lower edge curls upward and outward, so that its lower portion becomes concave. Higher up on the septum this concavity gives place to a compensatory convexity. The convexity generally is toward the spur. On the side of the delayed tooth a short basal spur indicates the enlarged premaxillary wing. The upper wisdom tooth may deform the septum posteriorly. Occasionally deformity of the septum is caused by asymmetry of one half of the palate. This asymmetry shows in the nasal notches anteriorly and in the choanae posteriorly and in the mouth. Such extensive asymmetry is due to unequal descent of the antra. After the teeth are fully erupted and in good line there remains no evidence of the disturbance caused by their delayed eruption except in the nose.

Trauma as well as delayed eruption of the incisor teeth can displace the premaxillary wings and distort the vomer groove resulting in spurs and causing deviations anteriorly and posteriorly. The best explanation for the slight anterior deviations which are found so constantly is some fault in the eruption of the incisor teeth. Abundant dissecting room findings prove that deviations so started may extend far back on the septum and become obstructive.

828 Beacon Street.

PRIMARY MELANOSIS OF THE PALATE. NASO-BUCCAL FISTULA OF RECENT SARCOMATOUS ORIGIN.

BY J. N. ROY, M.D., MONTRÉAL, CANADA.

It is well known that there exist strange freaks of nature which till now the most profound observers fail to explain. In medicine, numerous examples could be cited; among others, primary melanosis of the palate is, I think, worthy of note. The histologist could easily tell us what melanosis consists of, but he would find it very difficult indeed to explain how and why that melanosis became primarily localized in the palate. Normally the mucous membrane of the human palate is of a pale rose color and whenever pigment appears it is symptomatic of a pathological process.

A glance at comparative anatomy shows that, in general, the palate of animals is whitish in color, and the mucous membrane is undulated with numerous transverse furrows. However, the horse, the cow, the dog, the deer and the fox may normally have a dark palate. The maki, on the other hand, has always a pigmented palate.

The caprices of nature must puzzle the biologist and make him wonder what could be the physiological explanation of how a white palate and a dark palate could exist in the same race of animals.

If the physiology of animals is so capricious, it is not at all the same thing with human physiology. With man, melanosis of the palate has always a serious signification; either it is a precursory symptom of future disease, or it is a complication of an already existing tumour, or else it begins with the new-growth.

Medical literature is very poor in notes on this subject; I have been able to find only two other similar cases reported; therefore I have thought it might be of interest to record the present case, and I would draw special attention to the beginning of this condition which dates back twenty years, and to the naso-buccal fistula of recent sarcomatous origin.

In September, 1906, J. D., a blacksmith, 43 years of age, consulted me at the Hotel-Dieu. He related that in 1886, he remarked on the median raphé of the vault of the palate a small round spot of a diameter of about three millimeters. This spot was not elevated and caused no trouble. A year before he had slightly injured

1. Read before the Canadian Medical Association, Montreal, September, 1907.

his palate with the stem of a clay pipe. The hemorrhage was trifling and the wound healed quickly apparently leaving no traces. During the following twelve years, this spot increased in diameter to about six millimeters without, however, becoming prominent. The only symptom he noticed was slight roughness to the tongue on pressure. He consulted a doctor who prescribed gargles and applications of tincture of iodine. During the first twelve years, the melanosis was painless, but after the applications of iodine pain appeared. Pigmentation extended gradually to the surrounding parts, and the mucous membrane became granular and irregular. Hard dark masses with a hemorrhagic tendency began to form. Four years later, all the space between the dental arch of the superior maxilla was filled with melanotic granulations. The feeling of local irritation succeeded the pain caused by the applications of iodine, which were discontinued for that reason. The irritation disappeared temporarily when the patient rubbed the mass with his tongue. The last four years, the lesion became deeper. Till then the whole palate was in a state of melanosis. Then slowly there came a depression more marked on the left side. We can not trace at present any swelling antecedent to this depression. The palate then became rougher and furrows separated small masses which apparently were increasing in size. Early in September last, the patient while attempting to suck perceived the existence of a naso-buccal fistula. Neither hemorrhage nor suppuration were noted.

On examination it was remarked that the melanosis invaded the whole hard palate. Granulations of a brown and black color and of various sizes were scattered around. They were hard and showed no tendency to hemorrhage. The left side was greatly depressed and at the union of the anterior two thirds with the posterior third the probe passed into the left nasal cavity. Pain was felt only if irritating food was taken, or if cold air passed quickly through the fistula. There was a slight discharge and slight necrotic odor. A mass of granulation tissue at the orifice of the fistula prevented liquids from entering the nasal cavity.

The voice was nasal. Taste was dulled; no dysphagia, but loss of pharyngeal reflexes. Gums and cheeks showed no melanosis.

The patient has always been a great smoker, he used to chew tobacco and took no care of his teeth which are quite discolored. The upper teeth are healthy, except the first left molar which shows caries to the third degree. The third right molar has been extract-

ed. The second left lower molar has also been extracted. The other teeth are quite healthy.

An anterior rhinoscopic examination shows a hypertrophic rhinitis on the left side. The septum is depressible and oedematous, and a small cartilaginous spur prevents the perforation being seen. On the right side, the septum is in the same condition and there is hypertrophy of the inferior turbinals.

No history of epistaxis or nasal discharge.

Posterior rhinoscopy shows hypertrophy of the posterior ends of the lower and middle turbinals. The posterior end of the septum is tripled in diameter. A whitish infiltration appears to come from the middle part of the septum and gradually grows more marked as it extends backwards.

At this rhinological examination, I have not found any melanosis.

The pharynx is slightly congested, the larynx normal.

Diaphanoscopic examination shows the right antrum to be transparent and the pupil luminous. On the left side the cheek is darkened and the pupil obscured. By means of Mahu's method, I examined the capacity of the left antrum, and find it holds two cubic centimeters of water which returns from the left nostril quite clear.

Cervical lymphatic glands on left side appear slightly hypertrophied.

A close examination of the eyes shows they are normal.

The iris is of a clear grey color without any abnormal pigmentation.

Refraction is:

Right eye $90°+0.25$ V=1
Left eye $90°+0.25$ V=1

The patient has always resided in the country and has enjoyed excellent health. The only sicknesses he has had were pneumonia in 1888, and mild cystitis in 1894.

There is no history of tuberculosis or lues.

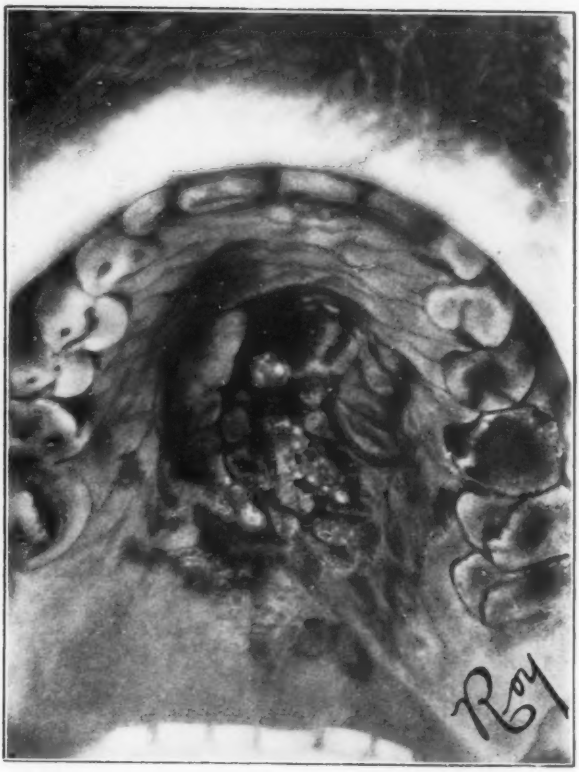
He has made use of alcohol without abuse.

Married for 11 years, he is the father of seven children. The two eldest died of convulsions in infancy. The others are in good health.

All his ancestors lived to a good old age.

There is no cancerous history.

His brothers and sisters are in excellent health.



Primary Melanosis of the Palate.

On inspection of the skin, there are no pigmented spots. The skin is normal except for a slight subicteric shade.

The examination of the different organs shows nothing abnormal.

Urine analysis shows a density of 1022. There is no sugar, or albumen, but a trace of urobilin.

Examination of the blood is of no special interest. There is nothing abnormal about its composition, and the number of red and white corpuscles varies but slightly from the normal. The presence of pigment in the blood was negative to a most minute examination.

Till now, the cause of this palatine perforation remained obscure.

Although it was impossible to trace lues in this patient, I prescribed large doses of Mercury and Iodide of Potassium awaiting the microscopic report on a small granulation removed for examination. Menthol and Boric Acil salve, and a mouthwash of Chlorate of Potassium completed the medical treatment. Hygienic treatment consisted in forbidding all irritating food and ordering cleansing of the teeth.

In this case we did not think it necessary to search for the Spirochaete Pallida of Schaudinn as it is generally admitted that this micro-organism is found only in primary and secondary lesions. As to the bacillus of rectilinear shape its presence is questioned in tertiary lesions.

My friends, Dr. St. Jacques, professor at Laval University, and Dr. Hingston, F. R. C. S.-Ed. returned the following report upon the small piece removed:

"On microscopic examination, the section appeared to measure about 5 millimeters in diameter. The epithelial layer still remains though varying in thickness in some parts.

"The cells of the deeper layers are in general well divided from the underlying parts; but in some places they appear to merge into the submucous tissue.

"The submucous space is filled with cells of irregular shape, various size and appearance. In some places they are very close to each other, in others they are well separated. Some of the cells are round, others very irregular. Mitosis not marked. The nuclei vary much in size and shape. The intercellular spaces are filled with granular substance, and here and there are seen free blood corpuscles. In one place, there are mucus glands altered in shape

but showing no signs of malignancy. Blood vessels are scarce but blood is seen in spaces without vessel-walls, typical of sarcoma. The cells of this mass are spread with no order whatever. Melanotic pigment is seen and is both intra and extracellular.

"Diagnosis: Melanotic sarcoma resembling melanotic endothelioma."

Seen again in November last, the patient said he was in the same state. The lesion appeared to have become deeper especially anteriorly. The vault of the palate was becoming denuded and in places small necrotic masses of bone were seen. There remained very little sensitiveness on the left side. He was very anxious to hear the result of the microscopic examination, and knowing that his condition was serious, he told me he would not consent to operative interference. He was given the necessary explanations, and the gravity of his state made clear. Operative measures were discussed together with the necessary mutilation, possible complications and probable recurrence. Having again refused the operation, he was given hygienic advice. A Resorcin gargle and Fowler's solution were prescribed.

He returned to the country where he now lives and came to see me in February and April. As yet there is no pain, but the lesion is rapidly increasing. The vault of the palate is becoming more denuded, and small sequestra become detached, thus increasing the size of the fistula. This atypical sarcoma so long indolent appears to be making up for lost time, and makes us foresee a fatal ending in the near future.

This case appears to be of interest from several points of view. First should we put the beginning of this melanosis back 21 years, and look upon it as consecutive to injury received at that period. We know that all cutaneous regions strongly or chronically irritated can become pigmented whether the irritation be physical, mechanical or pathological. Could that law be applied to our case? It would be difficult to admit it as the tissues of the human palate have not the property of producing melanotic pigment.

Should we call in a parasitic theory or attribute the cause of this melanosis to the melanin of the blood? Any hypothesis we could lay down would be most problematical for, we must admit, our knowledge on the etiology of this condition is extremely vague.

This neoplasm surely did not begin with the melanosis, as it is generally admitted that melanotic sarcoma has a very rapid growth. By the clinical history, we see that the pigmentation dates back 20

years, that the evolution was very slow, and that the symptoms were slight. Sixteen years after it had appeared, the mark was only six millimeters in diameter, and was on the level with mucous membrane of the palate. Perhaps the irritating action of the iodine caused the increase of the growth of the tumor in the breadth and depth. Perhaps it was mere coincidence. The naso-buccal fistula appeared September last, and since then has rapidly progressed. From these facts, I am inclined to think that this primary melanosis was benign in nature, and that in late years a neoplasm appeared in this location and became infiltrated with pigment and gave to this lesion an invading, destructive and openly malignant growth.

Dalbet affirms that all melanotic tumors arising elsewhere than in the eye and the skin are sarcomatous. The microscopic examination of tissue removed a year or two ago would have been of great interest. Perhaps at that moment we would have found an endothelial tumour. Pathologists do not agree on the histological transformation of endothelioma and sarcoma. Monod and Arthaud hold that sarcoma is an aggravated form of endothelioma. At present our tumor is certainly sarcomatous although in places an arrangement of cells resembling endothelioma are found.

What is the prognosis? We know that of sarcoma the melanotic variety is one of the most serious. Moreover when the growth invades the palatine vault and perforates it, the evolution is rapid and invariably has a fatal termination.

If operative measures had been attempted recurrence probably would have occurred on account of the growth of the lesions towards the nose and left antrum. We would also have had the risk of post-operative complications and considerable disfiguration.

In conclusion, I should like to remark how unusual this case is, presenting a primary melanosis of the palate without coexisting lesions of the eye or skin, a slow evolution of twenty years, and a recent rapid sarcomatous growth.

379 Rue St. Denis.

SUPPURATION IN THE TEMPORAL FOSSA.*

BY H. GIFFORD, M.D., OMAHA, NEB.

I happen to have seen two cases in which pus from the middle ear instetad of following the lines described in most text-books and breaking through on the outer surface of the mastoid, or into the digastric fossa, has penetrated into the temporal fossa.

One of these patients was a girl of seven years, who was sent to me with the history that a month before, the left ear began to pain and discharge. Some days later a swelling appeared above and in front of the ear. This was opened by the family physician, but the history does not state whether he found any pus or not. The swelling continued, however, and during the twenty-four hours before she came to me the lids of that side became oedematous. I found a rather anemic girl with the right ear normal, the left ear discharging non-foetid pus through some part of the drum head which could not be clearly made out on account of the swelling and adherent epithelium. Between the left ear and the eye the tissues along the zygomatic process were very much swollen, the swelling extending out into the eyelids. There was a small opening in the skin half way between the ear and the eye which was not discharging any pus; deep fluctuation could be felt. Tepm. 103°. No decided swelling nor tenderness over the mastoid. Under chloroform, two incisions were made down to the zygomatic process which was found to be bare; and much pus was evacuated; paracentesis was also made as the posterior part of the drum head seemed to be bulging. Symptoms improved for ten days, but then the temperature again rose to 103° and more swelling was apparent above the auricle. Incision here down to the bone evacuated more pus. Two days later the mastoid was opened and pus found in the antrum, but the cells were not involved to any extent; after this the recovery was uneventful except for a peculiar pemphigus-like inflammation of the auricle which subsided under lead and opium dressing.

The other patient was a boy whom I saw only at his home and the exact dates of whose affection I can not give. The main facts of the history, however, are as follows: At the age of ten years, in the course of an acute attack of inflammation of the left middle ear

*Read by invitation before the Western Section of the American Laryngological, Rhinological and Otological Society, Denver, Colo., February 16, 1907.

with the escape of pus from a small perforation in Shrapnell's membrane, he developed a large swelling with tenderness and very slight redness in the preauricular region, which kept increasing slowly with very little pain, for several days. I then, although no fluctuation could be made out, made an incision about an inch in front of the meatus, passing the knife into a depth of about an inch just above the upper margin of the zygomatic process. A few drops of pus escaped and the swelling gradually subsided and the discharge from the ear ceased. A year late, after catching cold, the left ear began to discharge again through the old perforation, and in a few days the tissues in front of the ear swelled to twice their ordinary thickness. This time, however, the swelling subsided under the use of hot applications and the discharge from the ear ceased on the same treatment as that used before, namely syringing with warm water and the instillation of a mixture of equal parts of saturated boric acid solution and 15 volume peroxide of hydrogen. The next winter was passed without any recurrence of ear trouble, and in the succeeding winter, the fourth after the first attack, the same sequence of events occurred. The boy caught cold, the left ear ached a little and then began to discharge through the opening in Shrapnell's membrane; the preauricular swelling appeared and slowly and steadily increased, until the soft parts seemed to be from two to three times their ordinary thickness. I incised the swellling to the depth of about an inch, but found no pus. The swellling went on with only moderate pain and slight fever for several days more when I again incised the swelling, this time passing the knife in past the zygomatic process until, at a depth of between two and three inches from the surface, the point struck the bone at the inner side of the temporal fossa. This cut evacuated about a drachm of non-foetid pus and the sypmtoms subsided as before. During the five years which followed he had two slight attacks of ear ache but by the prompt and vigorous use of hot water in the meatus his mother succeeded in checking the inflammation within a day or two, and since then, by scrupulous attention to the cleansing of the nasal passages and by taking large doses of aconite at the first sign of a cold, he has kept from having any further trouble for about five years; but I have no doubt that if he should allow another otitis media to get well started he would have a recurrence of the abscess in the temporal fossa.

Before finishing my remarks on this case I wish to state explicitly that at no time did the boy have any pain, tenderness, or swelling behind or above the ear.

Since my first introduction to this complication of middle ear disease, I have kept on the lookout for reports of similar cases in the literature, and have noted only the following:

1. Massier (*La Presse Oto-laryngologique Belge,* Jan., 1903, abst. in *Arch. f. Okrenheilk.*), reports two cases of abscess of the temporal fossa. One of these was of auricular origin.

2. Raoult (*Rev. hebd. laryngol.,* 1902, 42, abst. in *Archv. f. Ohrenheilk.*), reports a case of abscess of the temporal fossa in connection with chronic suppuration of the middle ear. A radical operation was performed but no disease of the mastoid was found; there was, however, a superficial necrosis of the anterior lower wall of the external meatus. The contents of the abscess were foetid.

3. Müller (*Fortsch. d. Med.* Aug. 1, 1904), reports case of Paul G., 19 years old; suppuration of the left middle ear after scarlatina; transient tenderness back of ear then swelling and slight fluctuation in front of ear. Incision found no pus till periostium of zygomatic process penetrated, then evacuation of 20 ccm. of thick pus; recovery.

4. Antonelli (*Rec. d'ophth,* May, 1905.) Girl aet. eleven months. Had the grip followed by free discharge of pus from the right ear. Two days after discharge appeared, tissues in front of ear began to swell, the infiltration progressing steadily forward until the whole zygomatic region and the lids of that side were involved. Antonelli saw the child about two weeks later and found a pasty swelling extending from the ear to the outer commissure of the eye lids. No fluctuation nor pain. After another week, Antonelli saw the child again with increased swelling of the side of the face and of the tissues of the upper lid. Fluctuation distinct over the lachrymal gland. An incision along the margin of the orbit evacuated a caseous mass which Antonelli considered to be the remains of the lachrymal gland, with considerable thick greenish foetid pus. A sound passed along bare bone to the apex of the orbit. A drain was introduced and the symptoms gradually subsided, though at the time of Antonelli's report the swelling of the cheek was not entirely gone and the ear was still discharging. No mention is made of any mastoid symptoms. Antonelli thinks the infection passed from the middle ear either through the Glaserian fissure or the incompletely closed petro-tympanic suture; thence between external pterygoid and the temporal sheath to the pterygo-maxillary fossa; thence through the spheno-maxillary fissure to the orbit.

In attempting to explain this complication it may be that, in cases like the first one in my report, the suppuration has simply

spread from the mastoid cells into the cells in the root of the zygomatic process and then has broken through either on the outer surface of the process as in Müller's case, or into the temporal fossa. But where mastoid symptoms have been entirely wanting and especially where, as in my second case, the complication has recurred repeatedly, it seems more likely that the zygomatic cells are infected directly from the middle ear, the occurrence being favored by some natural defect in the tympanic wall. We should remember that the zygomatic and squamous cells are originally and perhaps always an entirely separate system from the ordinary mastoid cells, sometimes attaining such a development that Schwalbe, in his Anatomie der Sinnes organe, speaks of the cavity in the root of the zygoma as the Antrum squamosum. So far as I know, no one has observed direct openings from the tympanum into the zygomatic cells, but Cholewa (*Deutsche med. Wchnschr.*, 49, 1888), in explaining the occurrence of abscesses above the temporal ridge, cites Zuckerkandle to the effect that out of 200 skulls, he found four with a direct opening from the posterior part of the tympanum into the lowest cells of the squamous portion of the bone; that is into cells which anatomically belong with the cells in the zygomatic root. It is possible also that incomplete closure of the petro-squamosal suture, as observed by Cholewa, may account for some of these cases.

With regard to the path taken by the pus or infection from the zygomatic root to the temporal fossa, most of the cuts which show cells depict quite a thick layer of compact bone between the cells and the fossa, but in a plate accompanying an article of Bezold's (*Arch. f. Ohrenheilk*, XIII, 1), a section of a temporal bone is shown in which one of the cells in the zygomatic root has only an exceedingly thin wall of bone to separate it from the fossa; and the infrequency with which the latter is involved is a strong indication that these zygomatic cells, on account of their separate anatomical origin are really but seldom involved in the infection of the real mastoid cells.

There is, of course, the possibility that the infection may have passed through the Glaserian fissure, and in Antonelli's case this or the incompletely closed petro-tympanic suture may quite possibly have been the route taken; but we should expect some marked interference with the action of the jaw if this had been the case with the other patients mentioned, and nothing of the sort was noticed in my cases nor in the others so far as I can determine.

Clinically, this complication is of comparatively benign significance. All the cases apparently have done well with no other treat-

ment than opening the abscess. In the therapeutics, the only point that I wish especially to urge is the necessity of sometimes going very deep in order to reach the abscess. We so seldom have to open abscesses in this region that, speaking from personal experience, I should say that there was a decided tendency for the aurist to get cold feet after having passed in the knife for an inch or so with no results. One can not help picturing the embarrassment which would be felt on cutting an artery at a depth of two or more inches from the surface; but as the last of my cases showed it is sometimes impossible to reach the pus without pushing the knife clear past the zygomatic process until it strikes the inner wall of the temporal fossa.

Adenoids. A. VINSONHALER, Little Rock, Ark. *Med. Herald*, February, 1903.

This paper discusses only the treatment of adenoids. Its author who has had experience in London hospitals, states that somnoform (a mixture of chloride and bromide of ethyl) has been by experience shown to be no better than chloride of ethyl, and not a bit safer. "Deaths are recorded against all of them." He holds the safest anesthetic is the gas ether sequence.

The paper advocates the use of adenoid forceps; the curette is not mentioned. According to Mr. Hewitt, the London anesthetist, the ideal position for removal of adenoids, is with the head turned to the right side, allowing blood to escape into the right cheek; but he adds that most operators cannot work when that position is used.

The reviewer, from long experience, has from the first found this position easy; and it is well known that Fletcher Ingals and others prefer it.

Mr. Hovell of London in every adenoid operation removes the posterior ends of the inferior turbinates, being led to do this by noticing a number of cases in which mouth breathing persisted after removal of the adenoids. EATON.

ACUTE OTITIS MEDIA TREATED BY THE OPSONIC INDEX METHOD. BY INVITATION.*

BY A. C. MAGRUDER, M. D., AND GERALD B. WEBB, M. D.,
COLORADO SPRINGS.

DR. MAGRUDER:—Mr. P. Merchant, age 42, consulted me November 26th, 1906, complaining of pain in right ear which had existed for three days and was so intense as to prevent sleep. He had had slight grippe symptoms for about a week. Patient not tubercular and has an absolutely clean bill of health.

Duration, three days, slightly impaired hearing, intense pain, no vertigo, auricle normal, mastoid not involved. Examination showed inflammatory condition of drum head with bulging of the posterior inferior quadrant.

Diagnosis—Acute Otitis Media. Advised paracentesis; refused. Forty-eight hours later perforation in the posterior inferior quadrant took place, and an abundant serous discharge ensued, although perforation was small, 1 m.m. Pain did not cease with appearance of discharge but was less. Microscopic examination showed almost pure culture of pneumococci. Saw patient every day and took unusual precautions to prevent extraneous infection. Examination of throat and nose showed redness and swelling around tube and a diffuse hypertrophic rhinitis. Eustachian tube was very large and wide open; even slight blowing of the nose caused air to pass out through the ear. For this reason especial attention was directed toward keeping nose and throat in as nearly aseptic condition as possible to prevent reinfection from that source. As long as discharge remainel serous the ear was wiped dry each day and powdered boracic acid insufflated. For two weeks, the discharge remained serous and then became yellow, thick, and stringy. The microscope showed no other infection than the original pneumococcus. On account of the stringy mucous appearing with the discharge, it was necessary to resort to irrigations to cleanse the ear. The perforation was enlarged to 3 m.m., and ear cleansed with warm boracic acid solution and twenty per cent Argyrol instilled.

This line of treatment was continued until December 24th, with no material decrease or increase of the discharge. During the time from December 10th to January 20th, I ran the entire gamut of

*Read by invitation before the Western Section of the American Laryngological, Rhinological and Otological Society, Denver, Colo., February 16, 1907.

treatment with Alphozone, Peroxide of Hydrogen, Argyrol, various astringents, etc., without any appreciable effect on discharge. Hearing was reduced to about one-half. Examination with probe revealed no denuded bone area and no bone dust found in sediment after washing. Microscope still showed pneumococcic infection only. The only thing I seemed able to do was to prevent other infection from complicating the case.

On January 20th, patient complained of intense headache over right temporal area extending over upper temporal ridge which directed my attention to, possibly, a circumscribed meningitis or brain abscess. Temperature normal and pulse seventy, as they had been through entire illness.

So far this case has presented nothing of unusual interest save my inability to stop the discharge; but during all this time it must be said that the patient, being a very busy man and keeping long hours in his store on account of the holiday season, did not quit his work and could not be kept quiet. He even made three short railroad trips to Cripple Creek, forty-five miles distant, during which time he was constantly engaged in matters pertaining to his business.

Patient had appointments in Chicago and New York which he must keep by February 1st, and in my despair, having heard Dr. Webb's lecture on the marked beneficial results obtained by Wright in these cases, I took the patient to Dr. Webb for opsonic treatment. This was January 20th, 1907, seven weeks after patient's first visit to me, and on the above date, I removed from the ear a specimen of the pus for Dr. Webb. His part of the work he will explain to you.

For two days subsequent to this visit to Doctor Webb, pain in right temporal region was very intense, and was controlled by ice bags and codein. Bowels were freely open with calomel and castor oil, and pain ceased. I concluded that the pain was not so much a part of the ear condition as it was an intestinal toxinemia.

January 23rd, I attempted to practice Baer's passive congestion treatment over ear, but failed on account of air passing in through Eustachian tube. Twenty per cent warm Argyrol solution was used in ear every day and a part of this passed into the throat.

January 24th, 3 p. m. Doctor Webb's treatment began. An hypodermic injection of fourteen million pneumococci.

January 25th. Discharge greater.

January 26th and 27th. Condition the same. Increased discharge.

January 28th. Discharge very much less and distinctly mucoid in appearance. Second injection of vaccine given today by Doctor Webb,—twenty million pneumococci.

January 29th. Ear nearly dry, no pus or mucus showing; only a slight amount of serum present. Ear wiped dry and boracic acid insufflated.

January 30th. The boracic acid insufflated the day before removed from ear in absolutely dry condition. This was wiped out and fresh powder insufflated.

February 1st. Dressings dry. Ear dry.

February 2nd. Patient left this morning for Chicago and the East.

CONCLUSIONS: *First*—An acute suppurative otitis media resisted treatment for sixty-one days.

Second—No mastoid involvement.

Third—Pneumococcic infection alone present.

Fourth—Six days from first injection of vaccine ear was absolutely free from pus and dry.

Fifth—While this opsonic work had been carried on for some time with marked success by Wright, in London, I am told by Doctor Webb that this is the first case of Suppurative Otitis Media so treated in Colorado, and it is his opinion that it is the first case so treated in the United States.

Sixth—If I do not improve my method of treatment in this class of patients Dr. Webb will get all of them away from me.

DR. WEBB.—Dr. Magruder's patient presented a condition of purulent discharge from the right ear, in which I found a pure culture of Fraenkel's pneumococcus. The patient stated that this discharge would soak six pieces of absorbent cotton daily. Complaint was also made of much headache the past few days. The discharge had lasted about 60 days. Cultures were taken in serum broth and plain agar. An excellent growth occurred with each.

January 21, 1907. Patient's opsonic index to this growth found to be .7.

January 24. Opsonic index again .7. Between these dates I had grown and standardized a vaccine according to Wright's methods, and an inoculation of 14 millions was now made.

January 28. Patient reported an increase of discharge for 24 hours after the inoculation. For two days following he had had

intense headache. Discharge has now almost disappeared. Inoculation of 20 million was made. Opsonic index on this day found to be 1.1.

January 30. Dr. Magruder reported to me that no trace of discharge remained.

It might be possible for inoculations to intensify headaches by increasing during the negative phase the inflammatory condition of the membranes of the middle ear. In this case, however, I do not think it responsible, because of the small size of the dose. Wright has shown that by using small doses the negative phase is largely avoided. The average dose of the pneumococcus is fifty (50) millions.

The pneumococcus will be found in the majority of middle ear inflammations following influenza, and Wright's methods have been eminently successful in effecting cures. The advantages of vaccines are exemplified in this case by increasing the patient's resistance, or opsonic index from .7 to 1.1 by the first dose. The second dose probably raised it still higher.

Since the communication of this report several similar cases have responded to the same method of treatment, resulting in complete cure. Dr. Magruder reports, ten months after above treatment, that there has been no return of the discharge.

Angiomatous Growth in a Case of Ozoena Originating During Pregnancy. STREIT. *Monat. fur. Ohrenheilkunde*, Aug. 1909.

In the beginning of her pregnancy a young woman, who was suffering from ozoena, noticed an increased secretion and gradually increasing obstruction on the right side. At about the fifth month, daily hemorrhages from this nostril began and continued until the end of pregnancy. After this the hemorrhages were less frequent, but more abundant. Examination showed the presence of a large red tumor, obstructing the entire right nostril. It proved to be an angiofibroma attached to the lower turbinated bone.

YANKAUER.

BLINDNESS DUE TO POST ETHMOIDAL EMPYEMA ACUTE. REPORT OF A CASE.

BY GREENFIELD SLUDER, MD., ST. LOUIS.

That blindness may be produced by disease of the post ethmoidal and sphenoidal sinuses, also of the Antrum of Highmore is well known. Onodi in a recent monograph "Der Sehnerv und die Nebenhœlen der Nase." (Alfred Holdr, Wien, 1907) has given the result of ten years' study of this subject and cited many cases, some with autopsy findings where the diseased process had extended to the optic nerve through necrosis of bone with meningitis, or through extension of infectious material by vascular channels with meningitis, and some producing blindness by pressure on the optic nerve. In Grafe's Archiv, 1907, Birsch-Hirschfeld cites additional cases. Posey and Paterson cite similar cases.

The optic nerve and chiasm lie above the post ethmoidal and sphenoidal sinuses with a layer of bone between, which is usually quite thin. Sometimes the post ethmoidal cells extend backward to the chiasm and lie above the sphenoidal cells so that this portion of the floor of the brain cavity is made by the post ethmoidal cells and not the sphenoidal cells. More often it is made by the sphenoidal cells which then extend forward to meet the post ethmoidal cells, they both being at the same height and each forming their part of the floor. The anatomy of the case I report must be of the first arrangement because the sphenoid was not involved. The blindness may have been from pressure, or toxins according to Birsch-Hirschfield, or both. This case seems to me of interest because of its acuteness, its dire consequences and the brilliant result of treatment.

W. H., male, 20 years of age, well developed, healthy and strong, applied to Dr. Luedde about 6:30 p. m. July 22, 1907, saying that "Three days ago the present trouble began with a sharp pain in right eye. 12 hours later a slight swelling appeared around this eye which lasted 12 hours. The pain continued until today. This morning at 7:30 both eyes became blind. The left eye remained blind about 25 minutes. The right eye can see direction of motions of hand 3 feet, vision of left eye 15-19 by artificial light. Examination showed the right upper meatus of the nose swollen shut and pale (oedematous). No pus could be discovered. Effort

was made to shrink away the swelling which was only in small part successful. He was placed in St. Luke's Hospital and the effort repeated at midnight, unsucessfully. It was repeated at 6 a. m. July 23, 1907, and followed by the discharge of about a teaspoonful of pus into the throat. Three hours later (9 a. m.) he reported to Dr. Luedde who found the vision, right eye 15-19, left eye 15-12. The discharge of pus continued intermittently, stopping in ten days. It always appeared between the middle and upped turbinates. Never did any appear in the recessus spheno-ethmoidalis. Four days from the beginning of the treatment the right upper meatus swelled shut again for a few hours (about six hours) the pain recurred to a slight degree and his vision in the right eye fell temporarily from 15-12 to 15-24 for this length of time. From the tenth day he remained normal in every way and continues so at present.

That the eye did not show changes in the fundus, Dr. Luedde thinks is explained by the central artery and vein of the retina not entering the optic nerve until about ½ in. from the globe and that the pressure clamped the nerve posterior to this point, that is, in the optic canal. I agree with this idea. Had this case gone on it would probably later have shown atrophy, descending. That the left eye should have been blind for a short time is probably explained by toxins as suggested by Birsch-Hirschfield in some of his cases. I am sorry that no examination of the pus was made.

3542 Washington Ave.

An Unusual Case of Scleroma of the Larynx. OTTO MEYER. *Monatschr. f. Ohrenh-*, May, 1906.

In addition to a beginning ozoena, this patient, who lived in a locality where scleroma is prevalent, had a nodular tumor as large as a bean on the right false cord. The rest of the larynx was normal. The tumor was removed with the galvano-cautery snare. Microscopic examination showed all the typical histological appearances of sceleroma. YANKAUER.

INFECTED CHOLESTEATOMA INVOLVING THE LABYRINTH AND ACCOMPANIED BY TYPICAL NYSTAGMUS.*

GEO. H. MATHEWSON, B. A., M. D., MONTREAL, QUE., CAN.

My object in presenting this case to your notice is to draw your attention to the importance of Nystagmus as a factor in differentiating between Suppurative Labyrinthine Disease and Cerebellar Abscess, for I well know that to most of you cholesteatoma of the temporal bone is no new thing. For those whose work lies chiefly along routine medical and surgical lines it may be well to state that cholesteatoma is simply a mass of epithelial debris, arranged in more or less definitely defined concentric layers between which cholesterin crystals are frequently found. It occurs in persons who have at some time had suppurative otitis media, with a resultant permanent perforation of the membrana tympani through which perforation the epidermal tissue of the external meatus has invaded the tympanic cavity and there proliferated and collected, forming a mass which may completely fill this cavity and the attic and mastoid antrum and erode the surrounding bone. It is worth noting that a dry uncontaminated Cholesteatoma rarely causes any trouble.

While for many years nystagmus has been recognized as one of the symptoms both of suppuration of the labyrinth and of cerebellar abscess, it is only of late that we have been able—thanks to the work of Neumann, Barany and others—to use the symptom as a means of establishing a differential diagnosis between these two conditions. Dizziness, nausea, vomiting and deafness may and generally do occur in both diseases, and severe occipital headache, which is generally accepted as strongly indicative of cerebellar abscess, may (according to Neumann), occur also in labyrinthine disease if this be complicated by localized meningitis. The workers in Politzer's Clinic discovered that the mode of occurrence of nystagmus is very different in the two diseases, and that in it we have our best means of making a differentiation. If, in a patient who is suffering from suppurative disease of the middle ear, nystagmus develops when he is made to rotate his eyes toward the healthy ear, and does not appear when the eyes are rotated in the opposite direction, we can assume positively the existence of suppurative disease of

* Read before the meeting of the Canada Medical Association, Montreal, Sept. 14, 1907.

the labyrinth. If, on the other hand, nystagmus develops when the eyes are rotated toward the diseased ear, and does not appear when they are rotated in the opposite direction, we may safely diagnose the presence of cerebellar abscess.

In progressive disease of the labyrinth the nystagmus disappears and hence is not seen in advanced disease when the membranous labyrinth is destroyed. Then, too, since by far the larger number of cerebellar abscesses arise from direct extension of suppurative disease of the labyrinth (90 per cent according to Alexander), we may have both forms of nystagmus in the same case. (As illustrative of the first point, I remember seeing a case of advanced labyrinthine disease in Dr. Politzer's Clinic, in which there was neither dizziness nor Nystagmus).

The history of my case is as follows:

On April 7th, 1907, C. B., male, a clerk by occupation, was brought to me complaining of dizziness, vomiting and weakness; he was quite clear mentally, and stated that he had been perfectly well until 22 days previously, when he went for a bath, and after diving noticed quite a severe pain in the left ear, which had never given him any trouble before. He applied hot poultices to the ear and three days later some pus came away from it. The pain was severe at times, but he kept at work most of the time. On Friday morning, April, 5th, he noticed that he was dizzy and that the pain in the ear had disappeared. He went to work, but noticed that he frequently saw objects double. After his supper he had a severe attack of vomiting. He felt dizzy lying in bed, no matter what position he assumed. On Saturday, he remained at home and during that day had two attacks of vomiting. On Sunday morning, he vomited again and seemed so sick that he was advised to consult me, which he did.

When seen by me about eleven o'clock on Sunday mroning, April 7th, i. e. two days after the onset of the dizziness, his condition was as follows:

He was a well nourished young man who looked very ill; his pulse was 66, and his temperature 97°—he had no pain, and his chief complaint was on account of the dizziness, which was so extreme that he was unable to walk alone, though with the help of a friend he could get along fairly well.

The right external auditory meatus was found to be of normal size. The drum membrane showed a small old perforation placed near the centre. Hearing normal on the right side. Examination

of the left meatus showed a great narrowing, due to a prolapse of the superior wall. Through this narrow opening I saw a wet reddish gray structure which I thought to be the lower part of the inflamed drum membrane, but which later proved to be the exposed face of the inflamed cholesteatoma. There was a small amount of pus in the meatus. There was no tenderness, redness nor swelling over the mastoid process. Tapping the cranium gave rise to no pain. There was absolute deafness on the left side.

On causing the patient to rotate his eyes to the left side there was no nystagmus, but when he was made to turn them to the right (or healthy side), there was a marked coarse nystagmus chiefly rotary. It was further noted that when the patient himself was rotated from his right to his left (being turned about rapidly three times), a marked rotary nystagmus was produced, while turning him in the opposite direction caused no Nystagmus.

The right fundus was normal, the left disc possibly somewhat blurred.

The patient was admitted to the General Hospital and I performed a radical mastoid operation the same afternoon. After the bone had been removed to a depth of about one-fourth of an inch, about 3i of creamy greenish fetid pus exuded, and it was then seen that the deeper part of the external meatus, the middle ear, and the mastoid were filled by a Cholesteatomatous mass. On removing this mass and the upper part of the posterior wall of the bony meatus, it was found that there was a carious opening in the bony labyrinth at a point about one-fourth of an inch above the fenestra ovale. The softened bone about this opening was gently curetted away. It was also seen that there was a carious opening about the size of a five cent piece in the roof of the antrum and attic, through which the dura could be seen. After curetting the walls of the tympanic orifice of the Eustachian tube to ensure the obliteration of that opening the membranous meatus was split longitudinally and the flaps forced against the walls of the bony cavity and held there by iodoform gauze and rubber drain, and the original skin incision behind the auricle completly sutured up. About a week later that part of the cavity not covered by the integumentary flaps was covered by skin grafts, and by the middle of June complete epidermisation of the cavity had taken place. It is interesting to note that immediately after the operation the dizziness was found to have disappeared.

56 Crescent St.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

CONJOINED SECTIONS ON LARYNGOLOGY AND OTOLOGY.

Meeting, May 29, 1907.

THOS. J. HARRIS, M.D., CHAIRMAN SECTION ON LARYNGOLOGY.
WENDELL C. PHILLIPS, M.D., CHAIRMAN SECTION ON OTOLOGY.
WENDELL C. PHILLIPS, M.D., PRESIDING.

Meningitis as an Intra-Cranial Complication in Diseases of the Accessory Sinuses. By L. A. COFFIN, M. D.

Meningitis as an Intra-Cranial Complication in Diseases of the Middle Ear. By ARNOLD KNAPP, M. D.

Statistics have shown that the meninges are first infected in nearly three-fourths of the cases in the posterior cranial fossa and in slightly over one-fourth of the cases in the middle cranial fossa. This is of importance in considering the possibility of operative treatment.

Forms of meningitis: Aside from serous meningitis, the classification of purulent meningitis into encapsulated, acute, progressive, and general, as suggested by Heine is the most satisfactory one.

Symptoms: Kernig's sign is the most constant. The value of lumbar puncture in diagnosis is discussed.

Prognosis: Less unfavorable than previously thought. Report of successfully operated-upon cases. Progress in the general variety of purulent meningitis however is absolutely bad.

Operative treatment: Broad exposure of the infected meningeal area if accessible, incision, drainage by introducing gauze tampons in the sub-ducal space.

As in general surgery advances in the treatment of peritonitis is due to the more thorough elimination of the primary focus, so in otitic meningitis our aim should be to better eradicate the primary focus in the temporal bone and that means in many cases the early recognition and proper surgical treatment of the labyrinthine suppuration. —Author's Abstract.

DISCUSSION.

PROF. G. KILLIAN, Freiburg, opening the discussion. Gentlemen.—Your esteemed president has asked me to open the discussion on Dr. Coffin's paper. I comply with pleasure, as I have had considerable experience with the complications following inflammation of the accessory sinuses of the nose. Aside from the three cases mentioned by Dr. Coffin which took a fatal turn after the frontal sinus operation, I have observed six other cases where the complications attending empyema of the accessory nasal sinuses were already well developed at the time when I was called to see the patients. The theoretical aspect of this question has engaged my mind for some time, especially in its anatomical relation. The results of my studies you will find in part in my atlas "The Accessory Sinuses of the Nose and their local relations to the surrounding organs." They have also been discussed and demonstrated on the occasions of the meetings of the Association of South-German Laryngologists in Heidelberg. I have arranged the respective paragraph in Paul Heymann's Manual of Laryngology and Rhinology, and have since then carefully followed up our literature on this subject.

It will be best, perhaps, to describe briefly those cases which have come under my observation.

Case 1. The patient suffered from chronic empyema of the frontal sinus on the left side, developed after a trauma received some months before. This case is described more in detail in my article on "Killian's radical operations of chronic frontal sinus empyema" in the *Archiv für Laryngologie*. Symptoms of acute inflammation developed in addition to the empyema of long standing. The facial expression was strangely altered, the walk reeling and uncertain, the pulse greatly retarded. On radical operation, I found the mucosa of the frontal sinus thickly covered with masses of fibrin which also permeated the mucous membrane. As the brain seemed to be involved, I removed the posterior wall of the frontal cavity and exposed the dura. It was sound, so that there seemed to be no indication for puncture of the frontal lobe for the present. In the course of a few days all symptoms of brain affection abated, and I reached the conclusion that it had been a case of slight serous meningitis. If I am correct, another such case has lately been recorded. As is well known, serous meningitis has often been noticed as a sequela of empyema of the middle ear. I take the liberty to mention, on this occasion, that the first recorded case of this nature

(Levy, *Zeitschrift für Ohrenheilkunde,* XXVI, 1894), was observed by me.

Cases 2 and 3. In two cases, I have observed suppurative meningitis directly following my radical operation of the frontal and ethmoid sinuses. In both cases, the operation had been successfully conducted without any mishap, and the wound showed every tendency toward healing by first intention. In spite of this, suppurative meningitis developed, resulting in death in the course of a few days. Autopsy showed that in Case 2 the wound was free from all infection. A portion of the middle turbinate in the region of the olfactory fissure, its lower edge situated at a distance of more than 1 cm. from the lamina cribrosa, showed a small trace of pus, and from this point a thin line of pus could be followed along the middle turbinate up toward the lamina cribrosa. This line of pus could be traced to the dura mater which showed signs of suppurative inflammation in small area and had caused the meningitis.

It was, then, a case of infection of a small area on the lower edge of the middle turbinate. How this infection developed is difficult to determine. The maxillary sinus had not yet been operated upon and contained very offensive pus. We had intended to perform this operation in a subsequent session. Two cotton tampons, which I had introduced prior to the operation and allowed to remain in place one day, had come in contact with the middle turbinate; and it is likely that the infectious pus from the maxillary sinus had been brought into close proximity to the point on the middle turbinate, thus causing infection.

In Case 3, which I operated shortly before my departure for the United States, I encountered empyema of the frontal, ethmoidal and maxillary sinuses on the left side, of fifteen years' duration. Polypi had also formed. The maxillary cavity was tapped from the mouth and syringed regularly, and the polypi removed. The patient (female) bled profusely even on slight provocation; therefore, when all acute symptoms of inflammation in the nose had disappeared, I operated only on the frontal cavity and the ethmoid bone, and found both much diseased. The posterior ethmoid cells were sound, so that I did not see any indication for the opening of the sphenoid cavity. Unfortunately this cavity was not carefully examined prior to the operation. For the first two days after the operation the patient suffered only slightly from headache and showed at times a slight increase in temperature; then meningitic symptoms developed suddenly, and the patient died two days later.

Post mortem section proved the wound entirely free from infection as in the other case. The very roomy sphenoid cavity showed chronic thickening of the mucous membrane and contained a glassy mucus. Only in its most posterior angle, directly below the sella turcica, did we find a small amount of pus. The suppurative inflammation continued from this point of the mucous membrane through the bone along the posterior side of the hypophysis to the inner layer of the dura mater. In this way meningitis had developed, and in spite of its short duration there had been a very marked discharge of a cloudy liquid. The liquor cerebro-spinalis being in abundance and under high pressure, had likewise clouded after only twenty-four hours, as was shown by lumbar punction. This case is very difficult to account for. As the maxillary cavity contained pus notwithstanding its irrigation, it is possible that on account of the position of the patient on her back particles of this pus had settled backward and downward, thus causing fresh infection of the chronically inflamed sphenoid cavity. It is to be assumed that the virulence of the bacteria present in this case had been greatly augmented on account of the different previous operative interventions and especially the frontal sinus operation itself. There is a possibility in this case that these changes had already begun to take place previous to the operation. It is a fact that toward the last the patient had complained of pains in the parietal and occipital regions, which must be attributed to the infection of the sphenoid cavity.

Case 4 presents the favorable termination of an abscess of the frontal lobe following empyema of the frontal sinus. This case occurred nearly nine years ago, but it has been reported in detail only in the Freiburger Association of Physicians. I had prepared a detailed description, but the manuscript was never completed because I was turned aside by bronchoscopy and other things. A number of analogous cases has been reported in recent literature, but by far the most of these died in spite of the operation. My patient was the second case where timely operative intervention brought about a cure. The first case is published by Denker in the *Archiv für Laryngologie*. Since then others have been recorded.

This was a case of frontal lobe abscess on the right side with extraordinarily typical forms of sclerosis. The indirect effect upon the inner capsule had caused hemiplegia of the left side. The patient was in a condition of complete coma and had Cheyne-Stokes' breathing when placed on the operating table. The posterior wall

of the frontal cavity was not perforated. When I removed this wall, I noticed a few drops of extra-dural pus. The inner upper area of the dura was covered with granulations of the size of a five-cent piece, and the center was perforated. When I increased the opening from this point, a large quantity of thick pus escaped from the frontal lobe. It seemed unaccountable that so large an amount of pus had been in the frontal lobe without causing perforation into the ventricle. The patient showed signs of improvement while yet on the operating table, and within eight weeks the brain abscess and external wound were healed. The most rigid examination and observation extending over a number of years failed to disclose the least psychic defect. The patient died seven years later of carcinoma of the liver. To my surprise, post mortem section revealed a cicatrix which affected only the extreme upper surface of the right frontal lobe where it had grown together with the wound. The volume of the lobe was the same as on the other side, so that I am constrained to assume that in this case only a small portion of the brain had undergone deterioration and that nearly all of the pus had collected by exudation.

Three other cases are characterized by general sepsis following extensive empyema of the accessory sinuses.

In Case 5, the accessory cavities had been diseased for years. High temperature, protrusion of the eyeballs, and acute hemorrhagic nephritis developed after intranasal operation. This was the patient's condition when I first saw her. Curettage of the frontal, ethmoid and sphenoid sinuses on both sides brought about no change. The patient died two days later.

Case 6 is analogous with the exception that influenza was added to a chronic infection of the accessory sinuses. Here, also, exophthalmos and hemorrhagic nephritis were evident. The patient was not operated upon, only treated with anti-streptococcic serum, and died likewise after a few days.

Case 7 seemed to me to have suffered from acute or subacute pansinusitis. High temperature and septic phenomena had developed. The patient was in the tenth month of pregnancy, and was delivered by means of instruments in the obstetrical clinic. Here, also, death ensued after a few days.

In Cases 5 and 6 we are justified, I think, in assuming a thrombosis in the sinus cavernosus which brought about the condition of general sepsis. Case 7 may have had a similar development.

Cases 8 and 9 were furnished by two patients with acute and subacute periostitis and osteomyelitis respectively, starting from the infected frontal sinus. In case 8 the patient suffered for years from empyema of the accessory sinuses and had sustained a fracture in the diseased frontal sinus region. The osteomyelitis was already in progress when she came under my observation. Notwithstanding several operations we were unable to check its course. The entire frontal bone rapidly became necrotic and meningitis was followed by death.

Case 9 could not be saved although four operations were performed. Almost the entire frontal bone had been resected, and in this way the process of infection in the frontal region had been checked, but it continued along the base of the skull toward the temporal fossa, penetrated the dura, reached the temporal lobe and caused an abscess which discharged into the ventricle, causing death. The patient had suffered from suppurative pansinusitis for ten years. Periostitis and osteomyelitis had developed after my frontal sinus operation, in the region of the most anterior and interior angle of the wound on the remaining portion of the processus frontalis of the upper maxilla. At this point, the virulent pus had settled between mucous membrane and epidermis. The infectious process developed slowly, the patient seemingly getting along well in the first few days.

This brief account of cases shows that disease of the nasal accessory sinuses may be followed by the same complications involving the brain as empyema of the middle ear. It cannot be doubted, however, that these complications as sequelae of empyema of the accessory nasal sinuses are much more rare. Still it must not be left out of consideration that patients may have died from meningitis of nasal origin when it had not been possible to determine this definitely.

Dr. Coffin has emphasized correctly that the manner in which infection travels from an accessory sinus to the brain has yet to be studied. I can furnish a small contribution to the anatomical side of this question. In the first place, I have studied very carefully the course of the blood-vessels and especially the veins in the mucous membrane of the accessory nasal sinuses, in the bone, and in the adjoining dura mater, with reference to the periostium. We succeeded in obtaining an excellent injection preparation which gave me the desired information. I have had drawings made of these preparations by our University drawing master, and demonstrated them a few years ago, in Heidelberg, but a detailed account

of this subject has not yet been completed. The results of my investigations were as follows:

In the mucous membrane of every accessory sinus we find a fine network of veins. These vessels are of largest caliber in the periosteal portion of the mucous membrane. The descending veins proceed for the most part through the mouth of the accessory cavity. However, the veins of the mucous membrane are in many places closely united with the veins of the bone and are continuous with them. In the bone we find a separate network of vessels. This can best be observed in thin bony plates as, for example, in the region of the lamina papyracea or in the walls between the cells of the ethmoid bone. If the bone is examined with a magnifying glass against the light, this fine network of vessels in its interior can plainly be seen. In the same manner as this network in the bone is connected with the mucous membrane of the accessory sinuses, it is also connected in many places with the veins of the dura mater or with the veins of the periosteum of the orbit or the periosteum of the entire nasal frame. In this way passages for the blood are everywhere provided, leading from the mucous membrane of the accessory sinuses through the medium of the veins in the bones to the periosteum on the other side to the dura. These passage ways are in many places long, complicated, fine, in other places they are short and wide. Sometimes there is a direct transition of a blood-passage from the mucous membrane of the accessory sinus to the dura mater. I consider these conditions exceedingly important, for they show that an extension of the inflammatory process from the accessory sinus mucosa to the dura is everywhere possible. The bone does not form an absolute barrier between the two. It restricts the relations of the blood vessels, but does not abolish them, just as a mountain range does not sever the connection between two countries, there being many mountain passes to establish communication.

I have not made a special study of the lymph passages. However, serial section made through the walls of an accessory sinus connected with the mucous membrane and the adjacent periosteum have shown that the bone is traversed only by blood vessels. It is to be assumed that the lymph passages take for the most part a like course with the blood vessels. Only in the region of the olfactory mucous membrane do we find broad lymph-sheaths which surround the branches of the olfactory nerve, and, as is well known, are directly connected with the arachnoidal space.

From a pathologico-anatomical point of view I would like to emphasize that septic processes are very often transmitted through the veins. Thrombosis takes place in them, bacteria lodge in the thrombi, these break up, and the process spreads in the vessels, leading to local and general complications. In considering the extent of inflammation from the mucous membrane of the accessory sinuses we must assume that such thrombo-phlebitic processes take place in the above described fine network of veins. The breaking-down of a single minute vein in the mucous membrane is sufficient to pass on the inflammation from this small area through the bone which in many places is very thin, into the corresponding vein of the dura. According to the intensity of this process will be its effect upon the cranium. If the inflammation takes a rapid course, then the liquor cerebro-spinalis is soon infected from the diseased dura and meningitis develops. If, however, the inflammation is less intense, if the dura, though diseased, does not permit the passage of the bacteria, then the dura, in the process of inflammation, comes in contact with the cortex and attaches itself to it, secondary relations between the vessels are formed in the inflammatory region, the pus-producing bacteria enter the brain substance and cause a brain abscess.

If the inflamed, diseased dura forms only the outer layer of a cerebral sinus as, for example, the sinus cavernosus, then a septic thrombus may form in the sinus which can easily lead to general sepsis or to meningitis.

Osteomyelitis holds an intermediate position. Here the infection progresses from the diseased accessory sinus at first only in the bone itself, and the process is chiefly confined to the diploe. Mostly, however, the secondary effects lead to the infection of the dura with the corresponding complications. In the case I have cited above, osteomyelitis began at the point where the bone was fractured.

These processes are to be explained in the first place by the action of the bacteria. Streptococci are generally found, but also staphylococci, pneumococci and others. As a rule these bacteria are of the same kind as those that have been lodging in the nose and its suppurating accessory sinuses for some time past, and we must assume that the patient had become used to the poisons produced by these bacteria, that is, there was a sufficient amount of anti-poison in the system. The patient was immune. This is probably the only reason why the inflammation had not reached the brain

in previous years. In simple inflammatory processes no bacteria are found with the microscope in the mucous membrane of the accessory sinus. If we assume that for any reason, a trauma (intranasal operation), a cold, a new infection, perhaps with a different kind of bacteria (influenza) the virulence of the bacteria already present has been greatly increased, or that more virulent germs have been introduced, then we can easily comprehend that a chronically inflamed mucous membrane is exposed to severe injury, especially as there are at first no corresponding anti-sera in the blood. If at any one place there is a greater degree of inflammation, it develops into direct necrosis of the tissues or into deterioration after previous permeation with fibrin. Any one or more minute veins in the mucous membrane at such a place transmits the disease with results such as we have just described. Whether this process develops rapidly or slowly is determined by the greater or less virulence of the bacteria.

We will, then, make some progress in this question only when we are enabled to extend our investigation not only to the kinds of bacteria that produce inflammation, but also to the degree of their virulence.

At present we have no adequate means of determining the virulence of streptococci. The results obtained with animals are not necessarily the same with men. However, the experiments so far made seem to indicate that there is a very great variation in the degree of virulence of streptococci in inflammations of the accessory nasal sinuses, and that even in inflammation accompanied by caries the virulence in considerably higher than in simple accessory nasal sinus empyema.

Time will not allow a more extensive discussion of the diagnostic side of these complications. For their therapy we are prepared by our experience in otology, for they are much more frequent in affections of the middle ear. Meningitis following accessory sinus operations takes so rapid a course that therapeutic measures are of no avail, not even Bier's treatment. Brain abscesses are often diagnosed too late.

In general sepsis, injections with anti-streptococcic serum may be attempted. In acute osteomyelitis and periostitis early radical measures are often successful. Luc has cured such a case. Perhaps Bier's treatment would be of value

Chronic empyema of the accessory sinuses should be operated upon in time, so as to prevent complications.

It is to be hoped that we will learn how to avoid such complications more and more. In the first place, there are the fatal consequences which result from a blunder of the operator. These must become rarer and rarer with increasing practice.

My clinical experience teaches me that it is a good plan to operate upon an infected maxillary sinus before attempting the frontal sinus operation to prevent subsequent infection.

In distinctly acute cases radical operation is not advisable; but if operative intervention is necessary, only the chisel should be used.

Also in acute exacerbations of chronic empyema of the frontal sinus it is better to wait with radical operation, if this can be done, until the acute symptoms have abated. If the operation must be performed, the wound should not be closed primarily, but at the earliest on the third day.

Since intra-nasal operations, especially in rapid succession, are likely to cause acute inflammation of the nasal mucous membrane and, therefore, to augment considerably the virulence of the existing bacteria, it is advisable to wait with radical operation until all signs of acute irritation have disappeared.

DR. GRUENING said that Dr. Knapp was in favor of early and complete operation, and in favor of operating even in the most desperate cases, for good results are often obtained even then. He agreed with Dr. Knapp fully on these points. He had with him a report from Mt. Sinai Hospital, taken from the last book covering a record of about fifteen months' work, showing 122 cases of mastoid disease operated upon during that time. There were only three cases of meningitis in all these; five cases of abscess of the brain (three of the tempero-sphenoidal lobe, and two of the cerebellum); and fourteen cases of thrombosis of the lateral sinus. There were fifteen deaths. If one remembers that the patients who come to Mt. Sinai Hospital are poor and neglected, and often arrive in a desperate condition, this mortality is really very small. When the diagnosis has been made, the patient is generally operated upon immediately. If lumbar puncture is required, the puncture is made when the patient is on the table, also the examination of the blood when needed, so that the lumbar puncture, the blood examination, and the operation are all performed at the same time if necessary.

There were more than three cases of meningitis. Why are only three so reported? Because they were so entered when they came into the hospital. If the condition of the patient is given by the most important symptom, then meningitis is the more dangerous

condition, for we can cure brain abscess more often than we can cure a general meningitis. Moreover, these cases of general meningitis are not only meningitis but are accompanied with general infection. In one instance, the meningitis was complicated by streptococcaemia, in another with pneumococcaemia; and one has to consider whether the meningitis is secondary to the streptococcaemia or. vice versa.

Dr. Knapp had not spoken of the ophthalmoscopic finding in meningitis. This is not important, and he did well not to mention it. He had published an account of a case of serous meningitis in which there was an optic neuritis. As regards the prognosis, it is immaterial whether or not that complication is present.

One condition which Dr. Gruening first observed many years ago is very interesting, i. e. mastoid disease complicated with herpes zoster in the course of the trigeminus. He told of a woman who had applied for treatment for otitis media. She had a very bad headache, high fever, and involvement of the second branch of the trigeminal nerve. He saw this case in consultation, and the patient died of meningitis. Later, he saw a second case of the same nature in Mt. Sinai Hospital, though he did not then connect the mastoid disease with the meningitis. This patient also died. Then he had a third case, and recalling these two which had been complicated with herpes, he said to his assistants that this patient would probably die. He thought that he had to deal with a meningitis, and that because the Gasserian ganglion was affected it was possible that the disease had already extended through the petrous portion—that there was probably a petrous bone with large air cells, and that probably the process had extended to the apex of the petrous portion, affecting the Gasserian ganglion, and thence had gone over to the dura, resulting in a meningitis. The patient died later of the meningitis, but unfortunately the Gasserian ganglion was not removed for examination. Dr. Gruening said that he had had five cases which presented this picture. In Dr. McEwen's book a fatal case of mastoid disease complicated with Herpes is reported. Dr. Gruening said that recently a report appeared from Dr. Brieger's clinic in Breslau containing similar observations. He thought that in this case the disease extended forward into the petrous portion, which was probably pneumatic instead of petrous.

Dr. Zabriskie said, in connection with the papers read tonight, he had had occasion to look up the records of the Manhattan Eye, Ear and Throat Hospital in regard to the relative frequency of

suppurative meningitis and brain abscess, and that for the past four years he had made autopsies on eleven cases, nine of which were meningitis and two abscess, but that these figures were of little value because they were so low. The question which had interested him most had been the factor which determined a brain abscess or meningitis in these cases of accessory sinus disease, and also the very interesting anatomical relation in regard to the lymphatics between the upper air passages, i. e., the nasal membrane, the mucous membranes lining the sinuses, in their relation to the dura. The only knowledge we have on the subject is the comparatively recent experimental work which was done by Cuneo and his students, and they have been able to show—how definitely, remains to be seen later—that the only direct lymphatic connection is between the dura and the olfactory bulb. They have shown fairly conclusively that the lymphatics of the dura are quite distinct from the sub-arachnoidal space following the sheaths of the nerves. That there is some lymphatic connection between the accessory sinuses and the dura, seems to be beyond doubt, but it still remains to be demonstrated. It is also a very interesting fact that we can have such a large proportion of sinus disease with relatively so few cases of meningitis, unless we consider another besides the direct continuity or exposure of the lymphatic or vascular layers to the infection. That factor, of course, must be the same as that which determines all infective processes.

Another most interesting question is the pathogenesis of brain abscess. Meningitis is, of course, easy enough to understand when we consider the vast lymphatic connections just spoken of, but brain abscess is another matter. Here we have an infective agent which experience has taught us practically always forms in that part of the brain nearest to the suppurating sinuses. Of course there is a certain proportion of cases where we have a direct continuity leading from the dura to the pia by means of adhesions, but there is also a certain number of cases in which this adhesion cannot be demonstrated, at least we have in our laboratory been unable to find it, and we are forced to either admit the almost impossible feat of the infective material passing directly through the cerebro-spinal fluid and lodging in the brain, or else we must search for an abnormal bridge by which the infective material can travel across the subarachnoid space, such as a small vessel. These vessels do occur abnormally, as we have demonstrated in the laboratory, but their relative frequency is yet to be determined.

DR. MYLES said that it had always been to him a mooted question how and why meningitis occurs. He had had thousands of operative cases of disease of the ethmoids and sphenoids, but had never yet had serious brain complications or brain symptoms directly associated, with the operative procedures. He had one case where he was about to operate on the frontal sinus—a man who had been under observation for some years previously, and upon whom he had often urged the necessity for operation. He appeared after a year's absence and consented to have the operation performed if he could be allowed a week's time in which to arrange his affairs. This was allowed, and later, before the time set for the operation, he received word that the man had died of meningitis. He had also had another case in which he had not operated. In an interesting case of grip, he operated upon an abscess on the inferior turbinal which had extended along the bone of the nose and out on the outside of the antrum. This was cut and the wound drained and packed. The wound healed, although the patient was more or less comatose for several weeks at that time. The general physician said that it was a meningeal disease, but there was no evidence of pus in the cells and the patient apparently recovered. About a year afterward he died of brain complications.

He claims that the phagocytic wall is the greatest barrier to meningitis in accessory sinus cases where the brain plates are disintegrating, and when this wall is disturbed it causes lymphatic absorption, or venous thrombosis, or some other means of transmitting the septic conditions to the meningeal cavity. Dr. Myles said that he always showed a great deal of respect for the meningeal membrane that passes down with the olfactory nerve, and attributes his freedom from fatalities to not disturbing that part of the ethmoid. In other words, he cuts off the middle turbinal, and always leaves the median wall of the ethmoid, especially in frontal sinus complications as he believes that thrombotic absorption or direct infection is probably the cause of some of the brain complications. In every operation that he has performed in this region he had always feared that death might ensue, but so far it has not occurred. He was always fearful of it, however, and always respected that part of the cribriform plate and crista galli region.

DR. DENCH said that about ten years ago he reported a case of otitic meningitis, in which he drained the subdural space over the tegmen tympani. The patient made a perfect recovery. Although he had operated on a number of cases of meningitis since this time all the others had terminated fatally. In the latter cases not only

had the subdural space been drained, but ventricular drainage had been employed in some cases supplementary to lumbar puncture. Aside from the first cases mentioned, all these cases had proved fatal. It was important to remember that, in these cases, infection usually occurs through the posterior aspect of the petrous pyramid, either through the aquaeductus vestibuli or the aquaeductus cochleae, or the infection may travel along the sheath of the auditory nerve. In the successful case mentioned by Dr. Dench, infection had taken place through the roof either of the antrum or tympanum, and had infected the meninges in the middle cranial fossa. From the success reported in decompressive operations for the relief of brain tumor, Dr. Dench was inclined to think a similar plan might be tried, with success, in cases of otitic meningitis. He was inclined to believe that the pressure would be best relieved by removing a large area of bone over the cerebellar fossa, incising the dura by a crucial incision, and then packing off the subdural space. In this way, the pressure symptoms would be relieved, and, at the same time, the subdural space would be drained. In the presence of any localized area of caries, indicating the route of infection, he was inclined to think that a decompressive operation would be more successful if the subdural space were drained below the tentorium rather than above it. If, however, a carious area was found at the time of the radical or mastoid operation, naturally this should be followed, and the dura exposed at the seat of the infection. Remembering that infections usually occur along the posterior aspect of the pyramid, particular attention should be paid to the condition of the bone in front of the lateral sinus, and any carious area found here, carefully followed. Ventricular drainage might be indicated in certain cases, but, at the present time, he believed that the simple decompressive operation, with incision of the dura and light packing of the subdural space along the incisions, should be the first step of the procedure. Ventricular drainage might be performed later, if necessary.

Dr. Coakley said that Dr. Coffin's paper directing attention to meningitis of nasal origin was most interesting. He thought that more cases of meningitis arise from purulent conditions of the nose than have heretofore been credited to that cause. Many children die of meningitis, and if these cases were examined at autopsy the point of infection would often be found to be from the nose or nasopharynx. Those men who are treating children realize that condition today as they did not a few years ago. He wished that Dr. Coffin had been able to arrange his cases so as to determine the

proportion of meningitis arising from intra-nasal operation as against the radical method, but that was probably impossible as yet.

Dr. Coakley himself felt that a rather complete external operation is much to be preferred to the intranasal operation. The latter is much more dangerous, especially in the frontal sinuses. Professor Killian's remarks would apply more particularly to intranasal operations followed by an external operation. That is because there is in many chronic cases a bacterium whose virulence is slight. He had met this difficulty in some experiments he had conducted this past winter. Streptococci taken from long-standing disease of the accessory cavities could not be made to grow on any ordinary media except blood agar; they could then be transferred to a second medium and would grow well. The intranasal work increases the virulence and quantity of these bacteria, and then if an external operation is performed one is much more liable to get infection. He had always previously considered it advantageous to do considerable intranasal work, and if that did not relieve the patient, to proceed with an external operation. All have had a certain number of septic cases result from the external operation.

Another point upon which Dr. Coffin had touched was that it makes considerable difference who does these operations. Many deaths follow sinus operations that are never reported, and there are good reasons why—the proper care was not observed. If these cases could be added to those reported by Dr. Coffin, the number of fatalities would be greatly increased. In many instances it was not the fault of the operator, but of his assistants. When working in the region of the ethmoid plate any undue force in sponging by the assistant can very easily perforate it. A trained assistant is as necessary as a trained operator.

Another point is the method of operating. In operating by the external route, in removing the ethmoidal cell labyrinth, if the instruments are not sharp enough the operator is liable to pull on the fibres of the olfactory nerve. Such traumatism is very dangerous. Sharp instruments should be used to cut off these fibres, so as not to endanger infecting the perineural lymph channels extending through the cribriform plate.

Dr. Coffin, in closing the discussion, thanked the gentlemen who had discussed his paper. With Dr. Coakley, he felt that the more he studied these cases in regard to intranasal work previous to radical operation, the more he was inclined to believe that we ought to do no intranasal work previous to the radical operation. If we adhere to this rule, we shall have less trouble.